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PLANT PATHOLOGY

BY

JOHN CHARLES WALKER
Professor of Plant Pathology
University of Wisconsin

NEW YORK TORONTO LONDON
McGRAW-HILL BOOK COMPANY, INC.
1950

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PLANT PATHOLOGY

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Plant pathology..

To My Wife

EDNA DIXON WALKER

PREFACE

The purpose of this text is to provide the basic facts whereby the student may be introduced to the scientific foundations of plant pathology. It is not a manual of disease descriptions nor a handy compendium of remedial measures. It presents rather comprehensive treatments of better known representative diseases which may serve as guides to the general principles underlying the science of plant pathology. Since the background of any science is basic to a full understanding of its present trends, a chapter on the history of plant pathology is included. Following the chapters on specific disease groups, consideration is given to environmental factors, host-parasite relations, and the methods of disease control.

In the preparation of this book the writer has profited greatly by the help and interest of his colleagues. Among those who have given their advice on various chapters and furnished illustrative materials are Deane Arny, M. P. Backus, J. G. Dickson, D. J. Hagedorn, P. E. Hoppe, James Johnson, G. W. Keitt, R. H. Larson, J. D. Moore, G. S. Pound, A. J. Riker, and M. A. Stahmann. Others who have provided photographs are Eubanks Carsner, John C. Dunegan, Donald Folsom, M. W. Gardner, G. F. Gravatt, R. E. Hartman, L. M. Hutchins, G. B. Ramsey, and C. Audrey Richards. The drawings were all copied from the original by Barbara Blumenfeld. I am deeply indebted to Eugene Herrling, who made many of the original photographs and carried out all of the final preparations of the illustrative material.

JOHN CHARLES WALKER

MADISON, WIS.
October, 1950

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CHAPTER 1

INTRODUCTION

Plant production is essential to the maintenance of life of man and animals upon this planet. Food, raiment, many luxuries, and some essential drugs are dependent directly or indirectly upon growing plants. Continuous change in crop requirements is brought about by shifts in population, in trends in consumer preference, and in relative costs of land, labor, and equipment. American agriculture is, indeed, dynamic and is increasingly dependent upon progress in underlying sciences.

Plant pathology is concerned with the health and productivity of growing plants. Disease losses are hazards which can be minimized only by a continuous process of research and education. Each change in variety of crop, each turn in the climatic cycle, and each regional shift in the growing of a crop brings new problems of health and disease. It is the responsibility of plant pathology to evaluate and solve new disease problems, to train research investigators and extension specialists, to brief agricultural educators and county agents in applied phases of the science, and to work out practical procedures which growers can adapt to their needs.

DEFINITION OF DISEASE IN PLANTS

For any variety or species of plant there is a set of environmental conditions which favors its optimum development. In nature, however, such an ideal situation seldom exists, and in consequence every variety of plant is subjected to the vicissitudes of the environment. When one or more factors in the environment become particularly unfavorable, the development of the plant is altered in some way, and in comparison with plants growing in the usual range of environment, it appears to have abnormal characteristics. Diseased plants are distinguished by changes in their structure or physiological processes which are brought about by unfavorable environment or by one or another parasitic agency. There is no clearly defined line of distinction between **normal** or **healthy plants** and **abnormal** or **diseased plants. Plant disease,** therefore, is, like many biological phenomena, difficult to circumscribe or define. We may, however, think of diseased plants as those which have become altered in their physiological and their morphological development to such a degree that signs of such effects are obvious. These external signs on the plant which are characteristic of a given disease are known as **symptoms.** Whether all deviations from the normal in structure and/or function constitute disease is largely a matter of definition and depends

in any case upon what one chooses to define as normal for the particular variety or species in question.

The development of man's concept of plant disease during the past 20 centuries is told in the next chapter on History of Plant Pathology. Recognition of abnormalities in plants goes back to the early historical records. In general they were attributed to the acts of supernatural beings, to vicissitudes of weather, to insects, or to parasitic seed plants. Only since the middle of the nineteenth century have parasitic microorganisms been accepted as causal agencies in plant disease. In the last two decades of the nineteenth century viruses were recognized as infectious causal agencies, and only since 1935 has considerable evidence been brought forward to show that viruses are nonliving but infectious proteins.

Since we now recognize that disease in plants may be brought about by unfavorable environment, or by infectious agencies which may or may not be living, it is well that we define at the outset certain terms which will be used repeatedly in this book.

DEFINITIONS AND TERMS

First of all it is well for the student to have a clear concept as to what constitutes the **cause of disease.** This is particularly true because the term is used rather loosely in much of the literature on plant pathology. It is relatively simple to conceive of one or more, or a combination of several, environmental factors becoming the cause of a disease when they alter the physiological processes and morphological development of the plant concerned. When a parasitic microorganism enters into the complex, as is the case in a large percentage of plant diseases, there is a common tendency to refer to the microorganism as the cause of the disease. Since we now know that variation in the environment is sometimes necessary to predispose the plant, *i.e.*, cause it to become susceptible to the microorganism, one can readily see that the microorganism is not the sole causal factor. Moreover we also recognize that the microorganism, also being a biological entity, is subject to the influence of environment, and it often, if not always, requires a certain range of favorable environment to produce infective organs and for such organs to infect the plant. After a microorganism has successfully infected a susceptible plant, the subsequent interaction of plant and microorganism which results in a disease of the former is also subject to environal influences which determine whether disease develops at all and, if so, at what rate and to what degree. It is evident that in the case of a disease in which a bacterium or fungus is concerned there are various other factors besides the latter which enter into the causal complex. Strictly speaking, therefore, it is not correct to refer to a given microorganism as the cause of a given disease, since this

implies that it is the sole cause. However, such usage is common in current literature on the subject. It has been suggested that the organism be referred to as the **incitant,** implying that it incites the disease under the influence of other factors. The term **causal organism** is also proper since it implies that the organism is a part of the causal complex.

Most causal organisms are parasites. In current usage a **parasite,** as applied to an organism, is one which subsists in whole or in part upon living tissue. Conversely, a **saprophyte** is an organism which subsists upon dead organic matter and inorganic materials. A **pathogen** is an agency which incites disease. While the term might be applied to any or all of the causal factors described above, it is practically always used to denote the living entity in the causal complex. The terms pathogen and parasite are not synonymous, however. In some cases organisms are parasitic without becoming causal factors in disease. In other cases the by-products of a strict saprophyte are an important part of the causal complex, and thus a saprophyte may indirectly become a pathogen. **Pathogenicity** is the property of a microorganism whereby it may become a part of the causal complex. **Pathogenesis** is the process or chain of events whereby disease development takes place.

A not uncommon habit among plant pathologists is to refer to the disease concerned by the scientific name of the causal organism. This is, of course, strictly incorrect. The student of plant pathology should at the beginning develop a habit of referring to the disease and to the causal organism as distinct entities.

Diseases of economic plants have usually acquired one or more **common names.** As a rule such names are descriptive of some important phase of the disease, *e.g.*, late blight of potato; wilt of tomato; black stem rust of wheat; clubroot of cabbage. Sometimes the generic name of the causal organism is used as a descriptive adjective in the common name, either because a sufficiently descriptive common word is not available or because it is necessary to distinguish between similar appearing diseases with different causal organisms. Thus wilt of tomato may be the result of the action of distinct causal organisms; hence, *Fusarium* wilt of tomato, *Verticillium* wilt of tomato, bacterial wilt of tomato. The *Rhizoctonia* disease of potato is another example of a case where the genus of the parasitic phase of the causal organism is used in the common name of the disease. This organism has a wide range of pathogenetic effects upon the potato plant. Under certain conditions basal stem cankers are formed, the result of which may be upward rolling of leaves, abnormal purpling of the foliage, deposition of starch in axillary buds of the stem to form aerial tubers. Quite apart from the disease on aerial parts of the plant, there are formed superficial black resting bodies (**sclerotia**) on the surface of

the potato tuber. Because the last symptom is a very obvious one, the disease is often referred to as black scurf. However, since the last term describes only one phase of a wide range of distinct disease reactions, the first term mentioned, *Rhizoctonia* disease, is also used.

The causal organism, if fully described, has been given a specific name within a given genus according to the accepted system of plant nomenclature. *Since the generic name and specific name are Latin words always printed in italics, they should always be underlined in any handwritten or typed manuscript.* This name of the organism is referred to as the **Latin binomial** of the organism. The species as originally described may be later transferred to another genus by the same or another investigator. In such a case the species name is retained unless it has been declared invalid for some legitimate reason. The name, or an abbreviation thereof, of the investigator who first described the species follows the binomial. If the species is referred by another investigator to another genus, unless the transfer is from a form genus to that of a perfect form, the name or abbreviation of the one making the original description is retained in parentheses immediately after the binomial, while the name or abbreviation of the one responsible for the current binomial follows.

Since there is not always agreement as to the proper genus to which a species belongs or as to whom the first authentic description of a species is properly attributed, there may be found more than one binomial for a given organism in current literature. On this account it is customary in a paper dealing with a given disease to give the complete binomial with the proper authority when the organism is first mentioned. This indicates clearly to the reader which authority the writer chooses to follow. Subsequently in the paper the authority may be omitted, and some writers prefer to make subsequent reference to the causal organism by using the common name of the disease as a descriptive phrase modifying "organism," "fungus," "bacterium," etc., *e.g.*, the crown-gall organism, the potato-late-blight organism, the potato-late-blight fungus, the fire-blight bacterium.

In certain cases usage has resulted in exceptions to the general rule, in that the same word is applied to the disease and to the causal organism. Most common examples are found in the rusts. The large group of diseases known as rusts acquired this name long before organisms were recognized as causal agencies, and the term came into use because it described a prominent characteristic of the diseases as a group. Likewise the organisms were known, and many were described and named, long before they were recognized as pathogenic agencies. Because of their characteristic color and the consistency of the spore mass, this group of organisms was also referred to as rusts. Thus the same word came into use for disease and

for pathogen long before their relations were fully understood. The same chronological history is true for the smuts. It is the more important, therefore, to connote whether the term is being used to apply to the disease or to the causal organism.

In the case of diseases in which parasitic organisms are a part of the causal complex the diseased plant is commonly referred to as the **host.** While usage has made this the most widely used term, objection has been raised that it does not imply the actual relation which ordinarily exists between the plant and the pathogen. The term **suscept** is a newer word coined to apply to the plant which is subject to a given disease brought about by a given causal complex.

The above terminology does not always strictly apply in the case of virus diseases. While the inanimate nature of viruses is not accepted in all quarters and practically nothing is known about their mode of increase or reproduction, they have above all in common with pathogenic microorganisms the property of being transmitted from plant to plant to become a part of the causal complex. While several schemes of nomenclature for plant viruses have been proposed, none has been fully accepted as yet. Therefore in current literature the student will find a great diversity among authors in method of reference to a given virus. They are not universally referred to as pathogens, and their disease-producing property is not commonly referred to as pathogenicity, largely because of the doubt that they are living entities. One system known as the Holmes system sets up a scheme of genera and species whereby each described virus is given a binomial. While this system has many admirable features, it is nonconformist in that it uses a system set up for living organisms to classify what may well be nonliving organic compounds, which cannot be defined on the bases used in the binomial system for living organisms. An earlier suggestion by Johnson is to give each virus described from a given suscept or host consecutive numbers, *e.g.*, potato virus 1, potato virus 2, cucumber virus 1, etc. A modification of the Johnson system, proposed by Smith, substitutes the generic name of the host for the common English name, *e.g.*, *Solanum* virus 1, *Solanum* virus 2, *Cucumis* virus 1, etc. Some writers follow one, some another, of these systems, while still others avoid all three systems and refer to the virus concerned by means of the disease with which it is associated, *e.g.*, common-tobacco-mosaic virus, potato-ring-spot virus, tobacco-ring-spot virus, spotted-wilt virus, potato-witches'-broom virus, etc.

When a pathogen is a part of the causal complex, the organism is first present in the environment in some overwintering (or oversummering) form. The overwintering stage may be in or on seed of the host, in perennial wild hosts, in debris of infected plants of the previous season, as

dormant spores, sclerotia, or other organs in the soil, in the form of an inhabitant of the soil flora, and in various other forms. Whatever this source, it becomes the **source of primary inoculum.** It may serve as the infective agent and thus become the **primary inoculum,** or it may multiply as a saprophyte (or as a parasite on a perennial host) to produce spores which become the primary inoculum. When the host becomes diseased, the organism commonly produces another crop of spores or infective bodies, which are known collectively as the **secondary inoculum** and serve to cause **secondary infection.** This last cycle may then be repeated several times during the current season.

The first step in the relation of the inoculum to the host consists of penetration. This may take place in one of a number of ways which are characteristic of the organism and are influenced sometimes by the environment. The various methods of penetration will be discussed in later chapters. It is important to recognize here that there is a distinction between penetration and infection. **Penetration** refers to initial invasion of the host by an organism. We know that many organisms penetrate cells of plants which are not susceptible and which do not respond with any sign of disease; moreover they die without proceeding beyond the stage of penetration. Thus we may have many cases of penetration without infection. **Infection** implies the establishment of the pathogen within the host following penetration. *It does not imply the production of disease symptoms.* There may be a long period between infection and appearance of disease, but ordinarily one follows rather closely upon the other. The interval is known as the **incubation period.** Just as penetration and infection are to be regarded as distinct, so infection and disease development are distinct. **Disease development** refers to the chain of events which occur between the time of infection and the complete expression of disease. In general, disease development is a protracted process which involves production of secondary inoculum and progression of external evidences of disturbance of the morphological and physiological processes of the host plant; these external evidences are referred to as **symptoms.** The chain of events in disease development is referred to as the **disease cycle.** While, when a pathogen is involved, the disease cycle is intimately associated with the organism, it is distinct from the **life cycle of the organism.** In the case of many causal organisms only part of the life cycle is passed in association with the host, the remainder being in association with dead host tissue, independently as a saprophyte, in association with an alternate host, or in association with an insect vector.

An **infectious organism** is one which may be transmitted from a diseased host plant to a healthy plant and which is capable of inciting the disease under favorable environment. An **infectious disease** is one in

which a transmissible causal organism is a part of the causal complex. Virus diseases are also usually referred to as infectious since the inciting agency can be transmitted from a diseased to a healthy plant and initiate the disease in the latter. In the light of present knowledge it is not proper to group them with infectious organisms, since there is no evidence that they reproduce themselves, whereas it is entirely possible if not probable that the infective entity acts as a catalyst which stimulates the formation of virus protein instead of normal host protein during the period of disease development.

Various **levels of parasitism** among microorganisms have long been recognized. In many cases two living organisms may live in association with one another, and in a sense each may be parasitic upon the other. However, they are not only nonpathogenic to one another but also are each essential to the other's development or at least each is beneficial to the other's development. This phenomenon is known as **symbiosis,** and the participating organisms are known as **symbionts.** Lichens are classic examples of symbiosis: a fungus and an alga living in close association with one another. The alga through its photosynthetic property converts kinetic energy of the sun's rays into potential energy of carbohydrates useful to its own development and essential to the life of the fungus. The latter through its property of producing hydrolytic enzymes breaks down materials in the substrate, making energy available and providing mineral and organic nutrients essential to itself and the alga. Another case of symbiosis is that of the nodule-forming bacteria in relation to their respective leguminous hosts. Here the bacterium penetrates and infects the root hair; the host responds by abnormal proliferation of cells to cause a gall, or root nodule. Up to this point the process might be considered pathogenic. However, the bacteria are capable of fixing the free nitrogen of the air and converting it into the essential nitrogenous compounds in their cells. Presumably, as the bacterial cells disintegrate, the nitrogenous compounds become available to the legume plant. Since higher plants are generally incapable of utilizing free nitrogen, an element essential to protein formation, this symbiotic phenomenon is a great economic source of available nitrogen. That symbiosis is a phase of parasitism and is very close to pathogenesis is shown by further study of the legume-bacterium phenomenon. All strains of the bacterium adapted to a given legume host are not equally efficient in nitrogen assimilation. In fact, some strains show little or no evidence of such a property. When such a strain is allowed to infect its legume host, nodules develop as usual, but in the absence of nitrogen assimilation the usual benefits to the host are lacking, and the latter shows certain disease symptoms when compared with uninoculated plants. Thus the legume bacterium is in this case not

a symbiont but a pathogen, and the parasitism involved results in pathogenesis rather than symbiosis.

Contiguous to parasitism but distinct from it is the phenomenon referred to herein as **epiphytism.** An **epiphyte** is a plant growing upon another plant, and, as the term is usually applied, it refers to cases where no parasitic or truly symbiotic relation is involved. Particularly in tropical regions, many higher plants grow on shrubs or trees, not in association with soil. They secure essential mineral nutrients from the dead bark tissue of the substrate plant but do not penetrate the living tissue of the latter and, apparently, have no beneficial or detrimental effect upon the latter. While some parasitic microorganisms are largely epiphytic, they are not considered as members of this group. Opposed to epiphytes are **endophytes,** which are contained entirely within the substrate plant. Such organisms may be either parasitic or symbiotic.

Having delineated symbiosis and epiphytism in relation to **parasitism,** we may now consider various grades of the last. After the relation of microorganisms to plant disease became fully recognized shortly after the middle of the nineteenth century, it soon became apparent that pathogenic organisms vary widely in the manner in which they bring about pathogenesis under the influence of favorable environment. Parasitism is a very complex phenomenon which is basic to the understanding of plant disease. Its various phases will be referred to repeatedly in subsequent chapters. Here it is essential only to explain the important terms used throughout the book. Some pathogens, so far as is shown, cannot complete their life cycles except in a parasitic relation to their respective hosts. They may persist as dormant spores away from the host, and such organs may germinate and produce secondary spores away from the host. Thus a part of the life cycle may proceed apart from the parasitic relation. Infection must occur, however, and parasitism obtain for the completion of the life cycle. Such organisms are known as **obligate parasites,** and they are found in various groups of the fungi; none are known among the bacteria. Viruses have properties in common with obligately parasitic organisms in that they increase in amount only in association with living suscepts. Common examples of obligate parasites are the rust fungi, the powdery-mildew fungi, the downy-mildew fungi, the crucifer-clubroot organism, the potato-black-wart fungus. **Obligate saprophytes** are those microorganisms which have no relation with living cells and secure their nutrients from dead organic tissue or from available inorganic materials. A large share of the pathogens, however, have parasitic and saprophytic phases of their life cycles. The fact that an organism is in part a saprophyte does not in any sense detract, necessarily, from its potentiality as a pathogen. Many organisms can live for indefinite periods

as saprophytes in pure culture or otherwise and still remain highly virulent pathogens. There are, however, many organisms which are in the main saprophytes but have weakly pathogenic properties which are ordinarily expressed only when particularly unfavorable conditions have predisposed the host in such a way that it is unusually susceptible and when such environment is particularly favorable to the expression of the weakly pathogenic properties of the organism. Such organisms are called **facultative parasites,** which term implies that they are ordinarily saprophytic but under certain conditions may become parasitic. Many damping-off organisms are typical facultative parasites. At the other end of the wide range of parasites that are not obligate parasites in the strictest sense are the **facultative saprophytes.** These organisms ordinarily have life cycles similar to the obligate parasites, in that they pass most of the cycle in association with the host and do not subsist as saprophytes. They may, however, subsist as saprophytes for part of their life cycle but do not complete their life cycle away from the host. Many of the smut fungi answer the requirements of this definition of a facultative saprophyte. It should be borne in mind, however, that there are no well-defined limits between facultative saprophytes and facultative parasites. It should be emphasized that they represent the extremes in the range of microorganisms which may have parasitic and saprophytic phases in their life cycles and *particularly that, as defined, the facultative saprophyte has a much higher potentiality as a pathogen than does the facultative parasite.*

Predisposition may be defined as the effect of one or more environal factors which makes a plant vulnerable to attack by a pathogen. It is a process which antedates penetration and infection. **Susceptibility** refers to a condition of a plant in which it is normally subject to attack by a given pathogen. The susceptibility of a given individual, variety, or species may be increased or decreased by environal factors. It may also be higher or lower as between individuals, varieties, or species because of differences in the inherited characteristics which affect susceptibility. **Resistance** may be regarded as the counterpart of susceptibility and also may be influenced in its expression by environal factors.

CLASSIFICATIONS OF PLANT DISEASES

Plant diseases may be grouped in various ways. The method of classification depends to a considerable extent upon the purpose of the treatment. In the chapter on History of Plant Pathology it will be seen that before the acceptance of microorganisms as causative agencies in plant disease (about 1850), many writers speculated more or less on possible causal factors, but they based their schemes of classification upon **effect** rather than upon **cause.** After 1850 books upon plant diseases grouped

them according to causal factors rather than according to effects or symptoms. Classification on the basis of cause has many advantages in a general text, one of the chief of which is that the most satisfactory approach to a basic understanding of disease is through an unraveling of the various causal factors and their interrelations, as well as their interaction with the host. For this reason such a scheme is used in this book.

In a manual of diseases of a certain group of plants, the consideration of or classification of diseases according to the host plant has decided advantages, and such a scheme is often used, *e.g.*, cereal diseases, truck-crop diseases, corn diseases, flax diseases, etc. In other treatises diseases are advantageously treated according to symptoms or effects, *e.g.*, root rots, powdery mildews, wilts, leaf blights, overgrowths, or hyperplastic diseases, hypoplastic diseases, necrotic diseases, etc.

Parasitic and virus diseases are often classified, in relation to their occurrence, into three groups: **endemic, epidemic,** or **epiphytotic,** and **sporadic.**

Endemic Diseases. When a disease is more or less constantly present from year to year in a moderate to severe form, it is classed as endemic. This is usually based on the fact that the pathogen or virus is well established and long persistent through its ability to survive from one crop season to the next in soil, on crop refuse, or in wild hosts. It is also implied that the environal conditions are usually favorable for inoculum to develop, infection to take place, and disease development to ensue. Diseases due to pathogens which persist for long periods in the soil, such as those of cabbage yellows and onion smut, are commonly endemic. When the infectious agent readily survives in prevalent wild hosts, as is the case with cucumber mosaic in milkweed or pokeweed plants, the disease is also usually endemic.

Epidemic, or Epiphytotic, Diseases. An epidemic disease is one which commonly occurs widely but periodically. The term "epidemic" is derived from a Greek word meaning "among the people" and in the strictest sense applies to diseases of man. The term "epiphytotic" has been coined to carry the same connotation with reference to plants. Epidemic diseases are often very responsive to variations in environment. The pathogen may be more or less constantly present, as in endemic diseases, but the environment favorable to development occurs only periodically. It is thus evident that the environment in such a case may well be the major determining causal factor. Epidemic diseases may also occur where the environment is usually favorable but where the pathogen is irregular in its occurrence. This may be because of the inability of the organism to survive always from one crop to the next. It may be dependent upon seed or other propagative parts of the host for

introduction. Insect vectors may be necessary for introduction or spread. Epidemics of black rot of crucifers are commonly dependent upon introduction of the pathogen with the seed. Late blight of potato is commonly epidemic in nature because of its sensitivity to fluctuation in temperature and humidity. Curly top, a virus disease of sugar beet in the Rocky Mountain states, is dependent upon the introduction of the virus by leaf hoppers which overwinter in more or less distant areas. If the winter reduces greatly the overwintering insect population, infection and spread are minor; if the conditions are the reverse, early and continued infection results in severe disease development.

Sporadic Diseases. Sporadic diseases in reality belong to the epidemic group. The term is applied to those which occur at very irregular intervals and locations and in relatively few instances.

It should be borne in mind that a given disease may be endemic in one region and epidemic in another. Late blight of potato is endemic in certain parts of northern Europe because the environment is practically always favorable at some time during the season. It is epidemic in the upper Mississippi valley and on the winter crop in the Gulf states, because some seasons are entirely unfavorable for its initiation and development.

DISEASES CLASSIFIED ACCORDING TO MAJOR CAUSAL AGENTS

There are two methods by which major disease classes may be set up according to cause. By one method, diseases are divided into two classes: infectious and noninfectious. In this case all diseases incited by microorganisms, parasitic phanerogams, nematodes, and viruses are regarded as infectious. However, it may be questioned whether certain of the insect-incited diseases are infectious, although they are parasitic. By the second method, diseases are divided into nonparasitic and parasitic classes. In this classification some authorities would place virus diseases in one class, and some would place them in the other. Thus these, like most or all methods of classification of biological entities, have obvious shortcomings at the outset. For purposes of utility, simplicity, and reasonable accuracy, the writer uses the latter scheme with the exception that the virus diseases are placed in a third distinct class.

I. Nonparasitic diseases.
   Diseases incited primarily by
   1. Low temperatures.
   2. High temperatures.
   3. Unfavorable oxygen relations.
   4. Unfavorable soil-moisture relations.
   5. Injurious atmospheric impurities.
   6. Lightning injury.

7. Mineral excesses.
8. Mineral deficiencies.

II. Parasitic diseases.

Diseases incited by

1. Bacteria.
2. Plasmodiophorales.
3. Phycomycetes.
4. Fungi Imperfecti.
5. Ascomycetes.
6. Basidiomycetes.
7. Phanerogams.
8. Animal parasites.
   a. Nematodes.
   b. Insects.

III. Virus diseases.

In the United States injuries brought about by insects belong in the field of economic entomology. This does not mean that the effects upon the hosts are not regarded as disease effects. Such a division of the field is an arbitrary one and is entirely justifiable for numerous reasons.

ECONOMIC SIGNIFICANCE OF PLANT DISEASES

It is not uncommon to find, in a relatively small locality, that the loss within a given crop in a given season from a single disease amounts to $100,000 or more. A very careful survey was made of some 6,000 acres selected at random from something over 100,000 acres of canning peas grown in Wisconsin in 1942. A very conservative estimate was that about 10 per cent of the crop was lost due to the inroads of one disease, *Aphanomyces* root rot, while another 10 per cent was lost due to the combined effect of all other diseases. Thus the yield of some 20,000 acres was nil. The cost of pea seed alone for 20,000 acres was approximately $20 per acre, or $400,000. The average gross return to the farmers concerned was about $75 per acre. Thus diseases caused altogether a reduction in return to growers of $1,500,000 in one crop in one state in one year. It may well be emphasized that this loss occurred almost entirely unnoticed by the public generally. There is no particular gain in adding up the annual loss in economic crops from disease which is sustained in this and in other countries. It is stupendous, but it would be difficult to determine accurately. Some is due to neglect; some is due to lack of education; much is due to lack of adequate information upon which to base corrective procedures.

It is also difficult to estimate how much loss is prevented annually by the application of remedial measures. Many crops are now dependent upon scientifically bred and carefully maintained varieties, or upon care-

fully grown seed stocks. Many crops are protected by carefully timed, scientifically devised fungicidal practices. As this is being written, a crop season is closing in the United States in which about 150,000,000 lb. of sulfur and 100,000,000 lb. of copper sulfate, in addition to various other fungicides, were used to protect crops from plant diseases. In spite of this, 10 million dollars' worth of tomatoes were lost in 1946 owing to one preventable disease in one portion of this country. In another part of the country, 2 million dollars was saved by watching the weather and withholding fungicidal applications because they were unnecessary.

Many such examples might be marshaled, but these are sufficient to show that plant-disease control is big business. Moreover, to serve the needs of crop protection, the science of plant pathology must continue to be a dynamic one. It can move forward only through research. Research rests upon understanding and initiative. A basic understanding of the principles of plant pathology is essential to the student in this field. The purpose of this book is to help inquisitive students to attain that goal.

CHAPTER 2

HISTORY OF PLANT PATHOLOGY

The earliest records of man reveal that as he began to depend upon cultivated food, feed, and fiber plants for his livelihood, the problems of crop losses, food shortages, and famine continually arose to plague him. We need only turn to the Old Testament (Gen. 41:23; I Kings 8:37; Amos 4:9; II Chron. 6:28; Deut. 28:22; Hag. 2:16–17) to find references in the history of the ancient Hebrews to blights and mildews of the cereal and vine crops upon which the people depended heavily. Greek philosophers, such as Theophrastus (84) (370 286 B.C.), noted the occurrence of crop maladies and speculated as to their cause and cure. Down through the Middle Ages we find that scholars were impressed by the appearance of plant diseases but were quite confused as to the factors which brought them about. Scientific experiments were not common, and the occasional keen observer of nature was hampered by the current custom of explaining natural phenomena by the process of deductive reasoning. Incorrect interpretations, based on unsound analysis of facts and influenced by superstition and religious dogmas, were passed from one generation to another until they became accepted as hard and fast laws of nature. At the close of the eighteenth century, about 150 years ago, there was no evidence that our modern concepts of the nature of disease inception in plants and the relation of environment to disease development had been adopted by those concerned with plant science and with plant culture.

DEVELOPMENT OF MYCOLOGY

Modern plant pathology did not develop alone; it grew along with and was influenced by related sciences. One of the most important of the allied fields was mycology. The fleshy fungi were thought by the Greeks and Romans to arise *de novo* from dead organic matter under the influence of the elements. This idea came naturally since all the early philosophers believed firmly in the idea of spontaneous generation of lower animals and plants. We shall find that the tenacity with which this theory was held until the middle of the nineteenth century was a dominant factor in the delay of the development of our present concepts of parasitism and disease.

In the sixteenth, seventeenth, and eighteenth centuries some prominent botanists were skeptical of spontaneous generation. Among these were Porta (64), Malpighi (56) (1628 1694), the pioneer in plant anatomy, and Jung (67) (1587 1657), Tournefort (86, 87) (1656 1708), and Jussieu (47) (1699–1777), pioneer plant systematists. On the other side were

such prominent and influential students of the plant world as Cesalpino (21) (1519–1603), another pioneer plant systematist, and Hooke (45) (1635–1703), an early microscopist. The discovery of bacteria (1683) by the Dutch draper, Leeuwenhoek (51), seems not to have influenced current thought on reproduction of lower organisms.

A contribution which stands out in this period, as we now look back upon it, is the work of Micheli (1679–1737). This Italian botanist studied both the higher and lower forms of fungi. In his "Nova plantarum genera" (16, 59) (1729), he described many new genera and illustrated their reproductive structures. He was the first to collect spores of fungi, sow them on organic substrate (nearly sterile pieces of melon), and watch the development of mycelium together with sporangia and spores characteristic of the species. He was convinced that fungi arose from their own spores. However, even these careful observations and the conclusions inductively arrived at were not accepted generally.

In 1748, Needham (17), an English clergyman and naturalist, upheld strongly the theory of spontaneous generation. To prove his point, he boiled meat in flasks, corked them, and set the containers away for a time, only to find that the contents had spoiled due to the microorganisms. In 1775, Spallanzani (79, 80), an Italian investigator, challenged Needham and set out to prove that decay of meat by bacteria and other lower forms of life could be prevented by heating the material in a flask which was sealed off in such a way as to exclude contamination from the air. His experiments supported this contention, but others failed to secure similar results consistently. These failures, we can now understand, were due to what would presently be regarded as faulty technique. This pioneer investigator likewise did not receive recognition and was successfully refuted for the time by his opponents.

The predominant attention of botanists from the time of Cesalpino (1519–1603) was directed toward naming and describing plants rather than searching into the phenomena whereby they grew and developed. By the turn of the eighteenth century the dogma of the constancy of species had become deeply ingrained in the minds of men, and this concept was fostered by the ecclesiasts, who found it to coincide well with the prevalent religious teachings. For 150 years or more, men had struggled to find a rational basis of naming and classifying plants. Linnaeus (1707–1778) was the leader in the eighteenth century who established the presently used Latin binomial system, his work culminating in the publication of "Species plantarum" in 1753 (55). This Swedish botanist had a wide influence. His students and followers were collectors and classifiers, but they were not experimenters. Micheli had been interested in morphology and taxonomy of the fungi, but he had also given attention to

their physiology and reproduction. He was ahead of his time. Linnaeus was a product of scholasticism and a firm believer in the dogma of constancy of species. He assented to the view that fungi arose through spontaneous generation.

While Linnaeus did not devote much time to the fungi, some of his followers specialized in this field. Prominent among these were Persoon (63) (1755–1837), who worked chiefly in France; Schweinitz (69) (1780–1834), in eastern United States; Fries (39) (1794–1878), in Sweden; Nees von Esenbeck (60) (1776–1858), in Germany; Léveillé (52–54) (1796–1870), in France. Persoon's "Synopsis methodica fungorum" (63), published in 1801, and Fries's "Systema mycologicum" (39), published in the years 1821 to 1832, became the foundation stones of the modern application of the Linnaean system to the taxonomy of the fungi. In the main, these men were concerned with the naming of fungi and with their classification, and they were not concerned with their reproduction and with their relation to plant disease. Although Persoon gave the name *Puccinia graminis* to the fungus associated with the black stem rust of wheat in 1794, he did not regard the fungus as the incitant of the disease. In 1818 he expressed the opinion that some fungi grew from spores, but that others arose by spontaneous generation.

In 1807, Prévost (65) observed germination of the spores of the wheat-bunt organism and conceived the idea that this organism penetrated the young wheat plant and was the actual cause of the disease. His revolutionary idea was refuted and did not gain favorable attention until 40 years later. Although predominant attention was given by most mycologists to taxonomy, there began about 1820 a renewed interest in the type of study that Micheli had undertaken 100 years earlier. Ehrenberg brought together known information on propagation of fungi and contributed further observations of his own on spore germination, as well as the first description of sexuality in fungi. Nees von Esenbeck repeated Micheli's experiment by sowing bread-mold spores on bread and later obtaining ripe sporangia. Dutrochet (1834) connected the sporophores of fleshy fungi with the subterranean mycelial strands which had been considered up to that time as distinct fungi. Trog (1837) confirmed earlier claims that fungi were disseminated by means of air-borne spores (67).

About this time there began to appear more discrete studies of the morphology and life history of fungi. The "Icones" of Corda (22), Bohemian botanist (1809–1849), published from 1837 to 1854, were the first result of the application of the compound microscope to the description of fungi. They were followed by the even more exacting and equally well illustrated studies of fungi by the French botanists, Louis René

Tulasne (1815–1885) and Charles Tulasne (1817–1884), whose works extended from about 1841 to 1865 (88 91). Contemporary with these men was Berkeley (1803 1889), the pioneer British mycologist.

PREMODERN PLANT PATHOLOGY

During the seventeenth century, plant diseases were increasing in importance in Europe with increase in population and in intensity of plant culture. Farmers, by and large, accepted them as inevitable concomitants of unfavorable soil and climate. Some growers, however, tried to do something to alleviate the damage, for we know that treatment of wheat seed with salt brine to control bunt began in that century (92). The association of the barberry with stem rust of grain had been recognized by farmers for centuries. It has been claimed by some that in 1660 farmers in the vicinity of Rouen, France, secured passage of a law which required destruction of the barberry bushes in that area. The authenticity of this report has never been firmly established. We are certain, however, that colonists took the barberry to New England with them and that wheat rust became increasingly destructive after 1660. A law was passed in Connecticut in 1726 which authorized town meetings to adopt barberry-eradication measures. Similar legislation was enacted in Massachusetts in 1755 and in Rhode Island in 1766. It should be emphasized that in the development of the control practices mentioned above there is no evidence that anyone had the present-day concept that many diseases are brought about by separate infective entities under the influence of favorable environment.

Theophrastus recorded his observations on the effects of various diseases of plants and in the main assumed that the basic causes lay in the vicissitudes of the weather. Pliny the Elder wrote upon the diseases of trees and of the cereal grains, but his work is little more than a translation of that of Theophrastus. Giambattista della Porta's discussion of diseases of plants (64), consisted largely of quotations from Theophrastus and Pliny, with some observations of his own. In 1705, the French systematist Tournefort (86) (1656–1708) classified plant diseases, dividing them into two major groups, one due to external causes, the other to internal causes. There followed a period of 150 years in which most writings on plant diseases consisted in descriptions of their appearances or symptoms and an arrangement of them into classes, genera, and species. The influence of the Linnaean binomial system was evident in each of these compilations. Adanson (1) (1763) in his work on "Familles des plantes" enumerated 23 species of plant disease, which were divided into two classes depending upon whether they were due to internal or external causes. The nature of the causes was, however, primarily a matter of speculation.

In Rozier's "Cour complet d'agriculture" (66) the list was expanded to 32 species. Plenck divided diseases into eight classes: external injuries, flux of juices, debility, cachexias, putrefactions, excrescences, monstrosities, sterilities (66). In 1773, Zallinger (101) (1731-1785) placed plant diseases in five classes: phlegmasiae (inflammatory diseases), paralyses or debilities, discharges and drainings, cachexia, or bad constitution of the body, chief defects of different organs. Fabricius (35), a Danish botanist who studied under Linnaeus and was for a time on the staff of the Royal Botanical Garden in Copenhagen, published a classification of plant diseases in 1774. He spent much time in Paris and evidently drew much of his material from Adanson's book. His treatise is an arrangement of diseases into classes, genera, and species on the basis of their symptoms. That Fabricius was not satisfied with current explanations of certain plant diseases is indicated by his statement that "certain it is that the cases and symptoms of smut can never be better explained than by assuming something organized to be the cause."

In 1755, Tillet (85) (1714-1791), director of the mint at Troyes, France, and amateur botanist and agriculturalist, published an important paper on wheat bunt. He found that opinion varied widely as to the cause of the disease—evil fogs, winds, excessive soil moisture, improper plant nutrients, soil texture, insects. In well-planned field experiments he tested the effect of dusting seed with the black mass contained in diseased kernels, and of treating such seed with water containing common salt and lime. He demonstrated that the disease was contagious, since dusted seed produced a much larger percentage of diseased plants at maturity than did undusted seed, while seed treated with salt and lime produced a lower percentage of diseased plants. Tillet did not recognize that the smut dust consisted of spores which were "seeds" of a parasitic fungus. He rather looked upon the spore dust as merely the carrier of a poisonous entity. However, he did establish for the first time that the disease was in some way contagious through the smut dust and that healthy seed produced only healthy plants. Furthermore, he demonstrated clearly that chemical treatment inhibited the contagious entity. In 1759, Count Francesco Ginanni (66) in Italy published a book on the same subject entitled "Trattato delle malattie del grano in erbo," in which he attributed the cause of bunt to minute insects. Another Frenchman, Tessier, working in the same period, published a paper entitled "Traité des maladies des grains," in which he attributed rust to a stoppage of the transpiration of the plant, caused by mists (83).

In 1766 an Italian, Giovanni Targioni-Tozzetti (66), put forward a theory that the rusts and smuts of wheat consisted of the vegetation of fungi arising under the epidermis of the plant. Fontana (38) (1730-1805)

an Italian physicist, published a paper the following year (1767) in which he expressed the view that the grain rust was a distinct parasitic entity. This new theory received some support, no doubt, but the weight of opinion was against it. In this same decade Anderson, an Englishman, wrote concerning the degeneration of potato varieties which we now know to be due to infectious viruses. In 1778 he published evidence to show that the malady was tuber-borne and likened it to smallpox in its infectiousness. Zallinger (101) in his book on plant diseases, "De morbis plantarum" (1773), upheld the prevalent concept that fungi found in association with plant-disease lesions were abnormal structures of the diseased plants.

BEGINNINGS OF MODERN PLANT PATHOLOGY

It is apparent that at the beginning of the nineteenth century when Persoon and, a little later, Fries were actively naming and compiling fungi without reference to their relation to plant disease, those who were writing about these maladies were still not cognizant of the true relation to them of microorganisms. The first investigator to bring forth the facts which clearly demonstrated the pathogenic nature of any microorganism was Prévost, a Swiss professor of philosophy at the Academy at Montauban, France. He studied the wheat-bunt disease for about 10 years beginning about 1797. He published in 1807 his epochal "Memoir on the Immediate Cause of Bunt or Smut of Wheat, and of Several Other Diseases of Plants, and on Preventives of Bunt" (65). After presenting an accurate description of the disease, he reported important results of experiments upon it. He studied and described the germination of the spores. He demonstrated, in confirmation of Tillet, that spores applied to the seed in some way resulted in infecting the plant. He studied the growth of the fungus within the growing wheat plant and its final development in the embryo where spores were formed, and where the life cycle of the fungus was completed. He showed that solutions containing copper sulfate prevented spore germination. He distinguished between fungicidal and fungistatic effects. From these facts he demonstrated the basis of control through seed treatment, a practice which had been carried on empirically and without any rational explanation for 100 years and more. The thorough hold of the theory of spontaneous generation on men's minds at this time was demonstrated by the fact that Prévost's results and interpretations were not accepted. A committee of the French Academy rejected his conclusions as unsound, and some 40 years had to elapse before others confirmed Prévost and pressed the adoption of the germ theory.

In the period from 1807 to 1845 the prevalent teachings are illustrated in the writings of Count Fillipo Ré, Franz Unger (1800–1870), and Franz

Julius Ferdinand Meyen (1804–1840). Ré was professor of botany and agriculture at the University of Modena, Italy. His treatise on the diseases of plants was published in 1807 (66), in the same year as the publication of Prévost's memoir. A second edition was published in 1817, and in 1849 Berkeley published an English translation of the latter in the *Gardener's Chronicle*, stating that it was the best book available on the subject. It is of interest to take more than casual note of Ré's book, because it portrays the increasing conflict of thought on the nature of plant disease in the beginning of the nineteenth century. Diseases were divided into classes and genera on the basis of their symptoms and their supposed causal factors. The plan was not fundamentally different from that of classifications presented in the last half of the previous century. The influence of current writings on the classification of human diseases is evident. We need mention here only the titles of the five classes: (*a*) diseases constantly sthenic (arising from excessive vigor, inflammatory); (*b*) diseases constantly asthenic (arising from debility); (*c*) diseases derived sometimes from the sthenic, sometimes from the asthenic state; (*d*) mechanical injuries, wounds; (*e*) indeterminate diseases. In the last class were those diseases which the author regarded as of uncertain origin. Among the diseases of that class were the rusts and smuts. Ré's comments on this group are quoted:

> I have thus designated those diseases whose origin is either entirely unknown, or deduced from observations contradictory in themselves, or from hypotheses which, however brilliant, have no real foundation. It is indeed a mortifying thing to see amongst these diseases precisely those which from the earliest times have been the subject of the studies of philosophers and that they are those which produce the greatest havoc in this country, while Nature refuses as yet to disclose to us the secret of their causes. Nevertheless, as I have said in my preliminary discourse, there are no vegetable diseases on which so much has been written by men of sound doctrine, of exactness of observation, and of careful judgment. . . .
>
> The principle opinions on the cause of rust, blight, melume, yellow blight, and carbuncle may be reduced to four. . . . The most generally received theory, first put forward in 1766, by the celebrated Dr. Giovanni Targioni-Tozzetti is, that these diseases consist in the vegetation of cryptogamic plants, arising under the epidermis of corn; and according to others, also on leaves and fruits of trees: and this has been confirmed by the microscopic observation of Fontana, Saussure, and more recently of Bauchs. . . .
>
> The opinion of Targioni has been latterly illustrated by several others, but I confess that I cannot yet bring myself to adhere to it. Some difficulties occur to me in the fact that notwithstanding the microscopical researches of Bauchs, others see differently also through the microscope. It appears also that he was unacquainted with the chemical and microscopical investigations of Girod-Chantrous, which have been passed over in silence by all those who have written on these diseases. . . .

Another difficulty in the explaining these difficulties by their being the result of cryptogamic, as it were intercutaneous vegetation, is the necessity of having recourse to principles which will not so readily be admitted by impartial persons. In the case of rust, for instance, some will have it that it is one plant, some that it is two different ones; and the manner in which they explain its appearance is precisely the same as that which those adopt who consider it an animal production. It is agreed that the seeds of these plants attack the Cerealia externally; and to show how this can be, they say they are minute enough to penetrate through the organic pores of the epidermis, establish themselves underneath it, and take root in the cellular tissue or parenchyma of the plant. Gautieri, on the other hand, in a memoir on the means of destroying rust in wheat, gives a totally different explanation of its mode of production, though founded also on the observations of Bauchs and Carradori. With all due honour to these great men, I confess that they leave me still in doubt, and I have therefore classed all these blights amongst diseases of which the cause is not determined.

If to all these opinions it is wished that I should add my own, I should commence by remarking that all agree in one point, that is, in the circumstances under which rust usually shows itself. I say, usually because some exceptions observed have occasioned those doubts which prevent me from laying down new hypotheses. Fields in low situations are generally the most subject to the disease, although in some years elevated spots have been more infected than low ones; so also fields well-manured or naturally rich have been the most affected. The milder or hotter the season the more these blights abound, especially in hot and moist years. Fields adjoining irrigated or rice grounds have most rusty plants. In little mountain valleys, much subject to fogs, blights abound. The greater the vigour of vegetation in the plant, the more it has to fear from them. All this would induce me to lay down that the cryptogamic plants, the minute insects, or the exudations, whether dry or not, are rather symptoms of the disease itself, which is a result of excessive vigour or over-repletion. And I am the more induced to believe it, as I have observed that weak trees and corn growing in poor soils are less liable to blights and rusts. It may also be that the transpiration is checked by the particles of wet (and for that they need not act as lenses but as mere obstacles), and that there follows a disorganization occasioned by the matter of transpiration itself, which might easily happen, considering that it is chiefly oxygen, and that facilities are thus given for the development of extraneous matter, vegetable, animal or whatever it may be.

The pressure of the parasitic theory is evident from the remarks of Ré, who evidently did not see the work of Prévost before the second edition of his book was published in 1817. The experimental evidence was lacking, except that presented by Prévost, to clinch the germ theory. The adherents of the autogenetic theory of disease still had the better of the argument.

Franz Unger was born in Germany but spent most of his life in Austria-Hungary. He was trained in medicine but in early mid-life turned to botany, becoming a professor of botany in Graz in 1835 and professor

of vegetable physiology in Vienna in 1849. In 1833, while serving as a medical officer in the Tirol, he published a book entitled "Die Exantheme der Pflanzen" (93). He believed that the fungus organisms associated with disease were endophytes but not parasites, that they had no independent existence, and that under certain environmental conditions every plant had the potentiality of producing them, in some way, from the waste material or exanthema of the plant. The influence of human pathology upon the interpretation of plant disease was still strong.

Meyen, a professor of botany in Berlin, was concerned primarily with plant anatomy and physiology, but he wrote a book on plant pathology which was published in 1841, in the year following his death (58). He followed Tournefort's system of classification of plant diseases. His erroneous concepts were characteristic of his period. He considered smuts of cereals to be noninfectious and to be due to a rotting of the sap brought about by improper fertilization of the soil. Rusts and mildews were likewise regarded as the by-products of disease due to environmental factors. He described the formation of the pustules of white rust on shepherd's-purse in a unique manner. He claimed that as the disease attacked the plant, cells of the latter enlarged and stretched out in some way toward the epidermis and cut off several spores in succession. Thus this incomplete picture of the development of sporangia as we now know it (p. 211) was misinterpreted to strengthen the autogenetic theory of disease.

About 1830, a few decades after the culture of potato as a food crop had expanded rapidly in Europe and North America, the late-blight disease appeared in both continents. It reached epidemic proportions by 1845. In that year it received much attention by writers in agricultural papers in Europe and the United States. The editorials and comments by contributors in the *Gardener's Chronicle* of 1845 are particularly enlightening to the student of plant pathology because they reveal better than any other existing evidence the trend of thought of laymen and scientists on the nature and cause of this destructive disease. While the association of the causal fungus with the blight of foliage and the rot of tubers was recognized by all who studied the disease closely, most writers held, in accord with the autogenetic theory of disease upheld by Unger (94) and by Meyen (58), that the fungus was the result rather than the cause of the disease. Von Martius, in Germany, was one of the first to distinguish the fungus (1842). He ascribed the disease to the effects of the fungus and published drawings of the latter. In 1845, Morren, director of the school of agriculture at Liége, Belgium, where the disease had devastated the potato fields, described the disease and the fungus associated with it. He conducted crude inoculation experiments with the fungus and con-

cluded that it was the major cause. He suggested the use of a mixture of lime, salt, and copper sulfate in suspension in water as a drench of the soil to reduce tuber rot. Had he applied this mixture to the foliage, he might have anticipated the discovery of Bordeaux mixture by 40 years. In the same year, Montagne, a French mycologist, collected the fungus in France, named it *Botrytis infestans*, and sent specimens to Berkeley in England. The spreading epidemic had not yet reached Berkeley's home in Northamptonshire, but it soon did. He confirmed Montagne's diagnosis and description but hedged on dogmatically upholding the idea that the fungus was the primary cause of the disease (7). English horticulturists, led by Lindley, editor of the *Gardener's Chronicle*, maintained that the unusual cool moist weather had so upset the normal growth of the potato that an internal breakdown of some sort followed. The fungus was universally present but entirely secondary, feeding upon and multiplying in the dead plant tissue. Lindley headed a commission to investigate the disease in Ireland, where acute distress and famine followed in the wake of the epidemic. After what Lindley and his associates thought to be a thorough investigation, the weather got the blame, and the fungus remained innocent, in spite of increasing evidence from some quarters, especially in America, that blight and rot occurred even where the weather was not abnormally cool or moist.

In 1846 Berkeley (8) took the stand that *Botrytis infestans* was the primary cause of the blight and tuber rot of potato. He maintained, further, that wheat bunt was incited by a fungus. In 1848 (9) he reported the potato fungus on *Datura stramonium L.*, on *Anthocercis*, and on tomato. He stated again with regard to the cause of the disease: "I have no more doubt that the malady is caused by the fungus than I have that many of the cereal diseases are caused by fungi."

What was lacking was experimental proof that *B. infestans* was the primary cause of the disease. Morren had provided some evidence, but the techniques of those days were still not adequate to refute completely the interpretations of the autogeneticists. Undoubtedly the supporters of the germ theory were increasing in number. Its general acceptance awaited only more clear-cut experimental evidence.

CONFIRMATION OF PREVOST'S WORK

In the decade of 1840 Louis René Tulasne (1815-1885) and Charles Tulasne (1817-1884), botanists at the Museum of Natural History in Paris, began extensive morphological studies of the fungi. Their excellently illustrated accounts of rusts, smuts, and Ascomycetes are classics in mycology. In 1847 (90), they published studies on the wheat-bunt fungus which confirmed the observations of Prévost with reference to spore

germination and relation of the fungus to the host plant. They extended their observations to several other smut organisms and to several rust fungi.

The work of the Tulasne brothers was followed in 1853 by a comprehensive paper on the fungi, including the smuts and rusts, by Anton De Bary (24) (1831–1888). Trained in medicine, De Bary early turned to botany as a career. This paper, published when he was 22 years of age, was dedicated to Alexander Braun, his professor of botany at Berlin, and a student of the algae. In De Bary's preface to his "Untersuchungen" in 1853 he wrote:

> The interest which the smut and rust diseases of plants, and their relation to the fungi constantly associated with them afford agriculturists and botanists, the incompleteness in many respects of our knowledge of these fungi, and the various controversies over their relation to the diseases concerned, as to whether they are causes or effects of these diseases, have prompted me to offer this book to the public.

He studied the development of several fungi in a critical manner. Taking first the corn smut, he traced the development of mycelium, sporiferous branches, and chlamydospores. He was impressed by the fact that the mycelium in the intercellular spaces was distinct from rather than a part of the host cell wall. In fact he pointed out that Unger had published the same observation in 1840, although the latter maintained that the fungus arose spontaneously as a by-product of disease. As the spores matured, he noted that the protoplasm of the mycelium passed into the spores and the hyphae became indistinct.

He saw that as the corn-smut gall began to develop, the host cells underwent continuous division up to about the time when spore formation was initiated. Then cell division slowed down gradually, and eventually the host tissue was broken down as the rapidly growing fungus drew nourishment from the cells. Three other smuts were studied, and the conclusion was drawn that all four species followed the same process in their development and differed only in morphological characteristics. He studied critically the development of pustules of white rusts of several hosts and again interpreted the mycelium and sporogenous hyphae as distinct from the host cell and not arising from the latter as maintained by Meyen.

De Bary examined critically several of the common rust fungi including that of black stem rust of cereals and that of rust of bean. Here he did not distinguish between summer spores (urediospores) and winter spores (teliospores) but described the latter and their formation accurately. Dimorphism of these species was not described until the next year by Tulasne (89). The aecial stages of rusts were still considered as separate

entities by mycologists, and De Bary was in agreement with that concept. He stated that although the opinion was universal that barberry was harmful to grain, there was no proof that the spores of *Aecidium* on barberry caused infection of cereals. Although he later was the first to prove this to be the fact (28), he was quite skeptical of the possibility in 1853.

Meyen had suggested that the pycnia which produced pycniospores in association with *Aecidium*, *Roestelia*, and other aecial rusts were male organs. De Bary discussed this possibility at length but was definite in his contention that no proof had been offered. He expressed hope that the matter of the relation of pycnia to aecia would be settled promptly since several workers were investigating the subject. As a matter of history, the question was not settled until Craigie (p. 406) presented evidence for heterothallism and explained the function of pycniospores in the rusts in 1927 (23).

Had De Bary published only the first two parts of his paper, the publication would have stood merely as a substantial contribution to the growing information on the fungi and their life histories and to the taxonomic relations of the forms considered. As to the taxonomy presented in Part II, it is evident that De Bary's attempts at classification were far from final. In Part III, however, he launched into the controversial field of the relation of fungi to plant disease.

Reviewing the opinions which had prevailed from the time of Theophrastus, he made it clear that the fungi associated with the diseases known as smuts and rusts were regarded by most farmers and botanists of De Bary's time as the effects of rather than the causes of disease. This, he explained, was because they saw these fungi appear simultaneously with the degeneration or failure of the host plants and were unable to study the formation of these degenerative lesions. Thus all manner of external factors were blamed as causes. As ideas changed, other theories arose to explain the association of the organisms with disease. Linnaeus (1767) considered the *Ustilago* spores as eggs of Infusoria. Later the similarity of smut and rust particles to fungus spores led him to include them among his fungus genera. Finally Persoon introduced a definite classification in which the genera *Aecidium*, *Uredo*, and *Puccinia* were set up.

De Bary wrote:

> After Persoon, few workers opposed the recognition of the smut particles as fungi; however, this did not prevent people from regarding them as products of disease in plants or from attributing the latter to entirely different causes.

Schleiden as late as 1850 still regarded the smut spores as diseased cell structures.

Unger, proceeding from the view that a disease is "a secondary, lower organism the elements of which lie hidden in a higher organism" declares . . . all smut fungi . . . are disease organisms. . . . Because of their similarity to fungi he says they are "imitations of normal plant forms." The species of smut and rust are designated by Fries, Wallroth and Meyen as fungi, but nevertheless as products of diseased conditions of the plants attacked. . . .

It must be determined, then, whether the smut fungi be parasites or products of diseased conditions, in other words, whether judged from the disturbances in the life of the plant which accompany their appearance, they are to be looked upon as cause or as effect. In the latter case they would be an insurmountable proof of spontaneous generation, but the facts upon which the advocates of this view rely have come partly from illusions and partly from a desire for theories and analogies which sometimes is really deplorable. Unger describes the smut (and rust) fungi as being formed from a matrix which has come by transudation from the plant cells into the intercellular spaces, an amorphous plasma from which the fungus spores are said to be constructed, as pus is formed from the exudate of inflamation in the animal body. Meyen's observations agree decidedly as to the presence of such a matrix, but I must denounce equally decidedly his statements concerning the formation of *Cystopus candidus* from cells changed by disease and concerning the origin of *Ustilago maydis*, etc., as being inexact and based upon illusion and imperfect preparation.

De Bary did not see Prévost's paper, but he knew of it through the reference to it by the Tulasnes. Without adding any new experimental results, he placed the weight of his judgment and his observational evidence squarely behind the claims of Prévost and challenged openly the contentions of Unger and of Meyen.

STATUS OF PLANT SCIENCE AT THE BEGINNING OF DE BARY–KÜHN PERIOD

In brief outline we have scanned some of the major workers and their contributions in mycology and plant pathology up to the time of De Bary in 1853. We have emphasized that throughout the eighteenth century and the first half of the nineteenth century the taxonomy of plants was paramount and the dogma of constancy of species had a strong influence upon the trend of botanical thought. Moreover, the theory of spontaneous generation of microscopic forms and of the fungi generally held sway. Parasitism was not an entirely new concept, for the parasitic phanerogams had been generally accepted as true parasites upon their hosts before this date, and De Bary in his first paper referred repeatedly to this fact in his argument for the acceptance of fungi as plant parasites.

It is important that we realize that other phases of botany also passed through transition stages as did mycology. The approach to natural science by experimentation and inductive reasoning was also affecting plant physiology and morphology.

Robert Hooke (1635–1703) was a British scientist and philosopher who by 1660 had greatly improved the compound microscope. He noted the cellular structure in charcoal and in cork. He published in 1667 a book under the title "Micrographia or Some Physiological Descriptions of Minute Bodies Made by Magnifying Glasses" (45). Two contemporaries of Hooke were Malpighi (1628–1694), Italian physician and anatomist, and Grew (1628–1711), British physician, who pursued studies of plant anatomy as an avocation. These two men presented results of their studies before the Royal Society of London in 1671, and both continued to publish in later years. Their work was largely exploratory and dealt with grosser features of anatomy. Malpighi published "Anatome plantarum" in 1675 (56). Grew published "The Anatomy of Plants" in 1682 (40). The important point of interest about these two men is that they advanced plant anatomy to a point which was not again exceeded for over 100 years.

Anton van Leeuwenhoek (1632–1723) (51) was contemporary with Malpighi and Grew. Not highly educated, he assiduously devoted his spare time to the preparation of superior lenses and made available to himself equipment capable of higher magnification than that employed by Malpighi and Grew. Born in Holland, where he carried on his work, he communicated his reports to the Royal Society of London. Besides discovering bacteria he also studied plant anatomy. His work as a whole was not so thorough as the studies of Malpighi and Grew, but with superior equipment he discerned some things which they overlooked, such as the crystals within certain plant cells.

Stephen Hales was the outstanding figure in plant physiology of the eighteenth century. His most quoted contribution is his book entitled "Vegetable Staticks" (41), which was published in 1724. Also in the eighteenth century were Kohlreuter (1733–1806), early plant hybridist, and the Dutch physician, Ingenhousz (1730–1799), who pioneered in the study of photosynthesis. He was the first to conceive that green plants took up carbon dioxide from the air and gave off oxygen. His work was contemporary with and helped by that of the French chemist, Lavoisier, who discovered oxygen and carbon dioxide.

In the first half of the nineteenth century, De Saussure (1767–1845), Swiss physiologist, advanced knowledge of the oxygen–carbon dioxide relation to plant nutrition and of the mineral nutrition of plants, while Dutrochet (1776–1847), also Swiss, studied the phenomenon of osmosis. Organic chemistry became a distinct science with the synthesis of urea in 1828. But it should be kept in mind that the advances of pioneers just mentioned were not very generally accepted. The pull to revert to deductive reasoning and to the philosophical rather than the experimental

scientific approach was still strong. Following De Saussure, Liebig became the dominant leader in plant nutrition and championed the use of mineral fertilizers in plant production. Nägeli, German physical chemist and plant morphologist, was one of the great German minds of his period. He pioneered the field of physiology of lower organisms.

During the first half of the nineteenth century there was revival of interest in anatomy. It was in this period that Schleiden and the zoologist Schwann formulated their cell theory (1830). In the field of plant anatomy von Mohl, Meyen, Unger, and Nägeli were all active. Robert Browne is generally credited with the first observation of the cell nucleus (1831). Dujarden, French zoologist, discovered the material within the cell we now refer to as protoplasm (1835). Purkinje, Bohemian biologist, was the first to use the term "protoplasm" (1839).

DISINFECTION, FERMENTATION, ANIMAL DISEASE

We have already stated that in so far as lower organisms were concerned, the prevailing concepts throughout the eighteenth century were that these organisms arose *de novo* from inanimate substrate. Needham, a Roman Catholic clergyman in England, undertook to prove that the theory of spontaneous generation was correct. In 1748 he published an account of his experiments, which consisted of boiling meat in flasks, corking them, and setting them away for a time. The contents eventually spoiled, and Needham, maintaining that all life had been destroyed by the heat, concluded that the organisms within the flasks arose *de novo*. Spallanzani (79, 80) challenged Needham and explained his results by arguing that unheated air had been allowed to contaminate the flasks. Others maintained that heated air was unfit for microorganisms. Spallanzani answered them by sterilizing air by other means. Nevertheless his views were not accepted. Appert (2), in France, stimulated by an offer of 20,000 francs by the French government during the Napoleonic wars for a means of preserving food, worked out a method published in 1810, which became the basis of the modern canning industry. He heated foods in sealed containers in boiling water, and they remained sterile. However, he did not try to explain the basis of his success. There continued much speculation on the matter with reference to both food preservation and fermentation. Schulze, Schwann, and Schröder and Dusch were workers who between 1836 and 1854 showed that sterilized vegetable infusions remained so if the air which passed into the container were first passed through sulfuric acid, cotton, or a heated tube.

Schwann (1837) and Cagniard-Latour (1838) discovered the yeast cell and maintained that fermentation was brought about by it. Berzelius, a Swedish chemist, on the other hand, in 1839, defended the mechanistic

explanation of fermentation, and he was followed in this support by Liebig, the German agricultural chemist. It was not until about 1860 that Pasteur furnished irrefutable evidence that microorganisms arise from preexisting living entities and that fermentation is a biological phenomenon rather than a purely chemical one. But it should be borne in mind that Pasteur's conclusions were not generally accepted for many years. Lister, the English surgeon, was, however, much impressed by Pasteur's work and with it as a background pioneered the field of antiseptic surgery (17).

The germ theory in relation to diseases of man and animals was not yet established. First final proof of the causal relation of the anthrax bacillus to the anthrax disease was published by Robert Koch (48) in Germany in 1876. The conclusions were soon confirmed by Pasteur. In 1882, Koch announced the poured-plate method of isolating bacteria by the use of gelatin as a liquefiable solid medium, which was a great improvement over the dilution culture method used by Pasteur. In the same year Koch announced his discovery of the tuberculosis organism.

THE PERIOD OF DE BARY AND KÜHN

In the three decades following 1853 De Bary became an outstanding figure in the botanical field. Much of his research was concerned with fungi and the relation of parasitic fungi to their hosts. He held important posts in several German universities. Beginning as a lecturer at Tübingen in 1853, he succeeded Nägeli at Freiburg in 1855. In 1867 he became professor of botany at Halle. In 1872, after Germany had taken Alsace-Lorraine from France, he became professor of botany and rector of the university at Strassburg. He remained in this post until his death in 1888 a few days before his fifty-seventh birthday.

It is significant that De Bary started his career as a teacher and an investigator in a decade destined to bristle with new biological viewpoints, activities, and discoveries. In 1858 Wallace and Darwin presented their ideas based on years of careful observation of variation in plants and animals and proposed their theory of evolution based on natural selection. In 1859 Darwin published his "Origin of Species," and the great struggle between the evolutionists and the adherents of the dogma of the constancy of species had begun. In the same period Sachs was becoming the outstanding leader in the beginnings of modern plant physiology.

The number of supporters of the germ theory increased markedly after 1850. The Tulasnes were active. In 1853 one of them published a paper on the powdery mildew of grape in which he took a definite stand that the *Oidium* was a distinct parasite (88). In 1854 Berkeley began the publication of "Vegetable Pathology," which ran as a serial in *Gardener's*

Chronicle through 1854, 1855, and part of 1856 (10). The majority of this treatise was taken up with accounts of nonparasitic maladies, and the system of classification followed more or less that of Ré. However, there was added a class under the name *Parasitae*, which included the rusts, smuts, mildews, and other diseases regarded as having a fungus as the primary cause. This was the first arrangement of plant diseases in which parasitic diseases were given even a minor place.

De Bary's outstanding leadership drew students from various countries in Europe and America. His laboratory became an active center of study in the new subject of parasitism. Many of his students became prominent leaders in the field of modern plant pathology. A few will be mentioned here to illustrate the caliber of these men and range of their nationalities. Woronin (1838–1903) studied with De Bary at Freiburg in 1859. He became the leading plant pathologist of his period in Russia. Brefeld (1839–1925) studied with De Bary in 1872–1873. He became an outstanding German leader in pure culture and life histories of fungi. In the field of plant pathology he is best known for his studies on the life histories of the cereal smuts Millardet (1838–1902) studied with De Bary at Freiburg. While professor of botany at Bordeaux (1876–1899) he was a leading student of the diseases of the vine and became most widely known for his discovery of Bordeaux mixture. Farlow (1844–1919) studied with De Bary at Freiburg in 1872. He spent his long active career as professor of cryptogamic botany at Harvard University, from which vantage point he exerted an outstanding leadership in the study of parasitic fungi from 1874 until his death. Ward (1854–1906) was one of De Bary's last students. He worked with him at Strassburg about 1880. He became the leading plant pathologist of England until his death at the age of 52.

While we cannot take space here to delineate the contributions from De Bary's laboratory, it is important to sketch briefly some of his major investigations, which served to clinch for all time the acceptance of the germ theory. Soon after the completion of his first paper he turned to a study of the downy-mildew fungi (*Peronosporaceae*). This became another thorough, pioneering study of the life cycles and the parasitic nature of this group of fungi. The first two papers were published in 1861 and 1863 (25, 27), the final one in 1881 (31). During this study he became involved with the most notable plant disease of the time, late blight of potato. The fungus being a close relative of the downy mildews, De Bary was well prepared to place on a sound basis the nature and life history of the organism, which up to that time was in a much confused state. He confirmed the work of Speerschneider (81, 82). He demonstrated the direct and indirect germination of the summer spores; the infection of tubers by

sporangia washed through the soil from the leaves; penetration of the fungus into the host and its subsequent development after infection; overwintering of the fungus as resting mycelium in the potato tuber. He disproved the claim of others that the fungus had resting spores; he placed the fungus in the new genus *Phytophthora*. His first paper on late blight appeared in 1861 (26), his last in 1876 (30).

Another matter still unsettled was the relation of rusts to their hosts, and the question of alternate hosts. The barberry had been suspected as an alternate host of the cereal black-stem-rust fungus for centuries, but no one had actually brought forth the proof. In 1865 (28) De Bary announced that he had successfully completed the life cycle of *Puccinia graminis* Pers., the black-stem-rust organism, by carrying it to the barberry and returning it to wheat. He reported the next year (1866) that he had found the alternate host of the leaf rust of rye, *P. rubigo-vera secale* (Eriks. & Henn.) Carleton, and that of the crown rust of oats, *P. coronata* (Pers.) Cda.

In his last major research paper (33), on the damping-off and vegetable-rotting fungus, *Sclerotinia sclerotiorum* (Lib.) DBy., he reported studies upon the physiology of parasitism. This pioneer work was among the first which were concerned with the nature of the interaction of host and parasite. He demonstrated that this fungus had a rotting action brought about by substances which diffused in advance of the mycelium.

De Bary also contributed to the advance of plant pathology through his compilations of current knowledge in the field of mycology. In 1866 he published a book entitled "Morphology and Physiology of Fungi, Lichens and Myxomycetes" (29). This appeared as the second volume of Hofmeister's "Handbook of Physiological Botany." His greatest compilation, "Comparative Morphology and Biology of the Fungi, Mycetozoa and Bacteria," was published in 1884, only 4 years before his death. Translated into English in 1887, it immediately became a valuable reference book in plant pathology (32). The thoroughness with which this volume was put together has scarcely been exceeded to the present day. It illustrated the masterful grasp of the subject which De Bary had attained, and in the chapters which are concerned with parasitic fungi it showed how completely the modern concept of parasitism had become established 30 years after he entered the field. "Lectures on Bacteria," a summary of "the present state of knowledge and opinion concerning the much discussed questions connected with bacteria," was published in 1886. An English translation appeared in 1887, and a second edition thereof was printed in 1898 (34).

Contemporary with De Bary and in many respects an equally important figure in the early development of modern plant pathology was Julius

Gotthelf Kühn (1825–1910). Born at Pulsnitz, near Dresden, Germany, his early background was associated with agriculture. His father was a small farmer, and at 16 Kühn became a farm manager, remaining in this profession until he was 30. He studied for a year in the agricultural academy at Poppelsdorf and for a year at Leipzig, where he was granted a doctor's degree in 1856. He lectured during the next year at the agricultural academy at Proskau and then returned to farm management on a large estate in Silesia. Here he published his epochal textbook in 1858, entitled "The Diseases of Cultivated Crops, Their Causes and Their Control" (49). This was the first text to be published in which the fungi were regarded as causal factors in plant diseases. In 1862 Kühn was called to the Chair in Agriculture at the University of Halle, where he began to build a college of agriculture in a major university. He remained at Halle until his death in 1910.

Kühn had a great influence on the development and application of control measures. In his textbook he recognized diseases due only to unfavorable environment and those due to parasitic organisms including insects, phanerogamic parasites, and cryptogamic parasites. He gave much attention to the improvement of seed-treatment methods for cereals. He published some 70 papers on plant diseases.

While Prévost had completed most of the steps necessary for modern acceptance of the bunt fungus as the major cause of wheat bunt, he did not determine how the fungus penetrated the host. Neither the Tulasnes nor De Bary added any new information on this subject, although De Bary eventually worked out the penetration of rusts and of the potato-late-blight fungus. It was Kühn who first demonstrated the entrance of the mycelium of the bunt fungus into the wheat seedling and followed its development in the host from that point to the formation of spores in the wheat kernel.

Michael Stephanovitch Woronin (1838–1903) was a productive worker in the field of plant pathology from the time of his association with De Bary at Freiburg in 1858–1859. His early papers dealt with chytrids and with sunflower rust. The severity of the clubroot disease of cabbage in the market gardens of St. Petersburg attracted his attention about 1870 (p. 155). This disease had been troublesome in Europe for centuries, but nothing was known of its cause. Woronin discovered by careful study the unusual type of parasite, new to science, which he placed among the slime molds. Still it was sufficiently different from the saprophytic slime molds to warrant the erection of a new order, family, genus, and species. The organism was named *Plasmodiphora brassicae*. A preliminary report in 1874 was followed by a full report in Russian in 1877, the paper being published in German in 1878 (100). This was the first description

of a disease incited by a parasitic organism closely related to the saprophytic slime molds. In the same paper he described a chytrid, *Chytridium brassicae*, parasitic on the roots of cabbage. Woronin maintained a lifelong interest in the fruit-rotting fungi belonging to the genus *Sclerotinia* and published several papers on them.

Soon after the appearance of Kühn's textbook, there appeared one by Willkomm (98) in 1866 on diseases of trees and one by Hallier in 1868, entitled "The Diseases of Cultivated Plants" (42). The latter followed the same plan as the book by Kühn. In 1874 appeared a text entitled "Handbook of Plant Diseases" by Sorauer (1839–1916) (78). This book is of special significance since it has passed through several editions up to the present time. In the first edition nearly as many pages were devoted to nonparasitic diseases as to parasitic diseases. In fact, Sorauer looked with concern upon the current trend of emphasizing fungus parasites and their life histories and neglecting the part of the host plant and the interaction of host and parasite in development of parasitic disease. He is one of the first to emphasize the predisposition of the host to disease by the influence upon it of preinfection environment.

Oskar Brefeld (1839 1925) had early association with De Bary in his mycological studies and conducted much research on the fungi extending well into the twentieth century. He pioneered the development of pure culture technique for the fungi. He published rather full descriptions of his methods in 1875 (11) with additional reports in 1881 (12) and in 1883 (13). His first interests at this time were in studying the complete life cycles of saprophytic fungi. His methods were gradually taken over by others and became a basis for present-day research in plant pathology, along with improvements introduced by Koch, Petri, and others. Brefeld turned his skill later to the culture of the smut fungi, and beginning about 1880, he continued to study this group for some 30 years. He did more than any one investigator to work out the life cycles of the cereal smuts (14, 15).

Another contemporary of De Bary and Kühn who contributed greatly to the field of plant pathology was Robert Hartig (1839-1901). He was trained in forestry and was professor of botany at the forestry academy at Eberswalde from 1866 to 1876. He was professor of botany at the forestry school in Aschaffenburg from 1876 to 1878, and in the latter year he transferred to Munich as professor of botany in the school of forestry and became director of the Royal Forestry Experiment Station. He became the outstanding leader in forest pathology of his generation. He contributed numerous reports on diseases of forest trees and on timber decay. An enthusiastic teacher, he was the author of two important texts in the field, "Important Diseases of Forest Trees" (1874) (43) and "Text-

book of Tree Diseases" (1882) (44). The latter was revised in 1889 and was translated into English, French, and Russian.

BACTERIAL DISEASES OF PLANTS

Woronin described bacteria in the root nodules of leguminous plants in 1866 (99). Burrill of the University of Illinois was the first to relate a bacterium to the cause of a plant disease. In reports extending from 1878 to 1884 (18–20) he showed that the fire blight of pear was incited by bacterial organism. It is of interest to note that the first bacterial disease of plants was demonstrated only two years after Koch had finally demonstrated the first bacterial disease of animals. In 1883 Wakker (95, 96), in Holland, published results of a thorough investigation of the yellow slime disease of hyacinth, which was shown to be incited by a bacterial organism. Arthur, working at the New York Experiment Station, in articles published from 1885 to 1887 (3, 4), confirmed Burrill's work. He isolated the organism in pure culture, using the newly developed plate culture technique of Koch, and fully demonstrated the pathogenicity of the bacterium. Savastano (68) demonstrated in 1887 that a bacterium incited the knot disease of olive. Erwin F. Smith began to study bacterial diseases of plants soon after 1890. His first attention was directed toward the bacterial wilt of cucurbits. His first report was in 1893, and a major paper was published in 1895 (72). He took up in succession the brown rot of solanaceous plants (73) (p. 126) and the black rot of crucifers (74), the latter of which had been first described by Pammel (61, 62) in 1893 (p. 122).

It is of significance that the leadership and most of the important work on bacterial diseases of plants from 1878 to 1900 and even later was found in the United States. In fact there was a distinct air of skepticism on the part of European botanists concerning the importance of bacteria as plant pathogens. When De Bary published his "Comparative Morphology" in 1884 (32), he was aware of Wakker's work on the hyacinth disease but apparently did not know of Burrill's work on fire blight. In the section on bacteria he devoted one short paragraph to those parasitic on plants. He pointed out that they had "scarcely ever been observed. . . . One reason for this may be that the parts of plants have usually an acid reaction." In "Lectures on Bacteria" (1887) (34) 2 of the 146 pages were devoted to bacteria parasitic on plants. The works of Wakker, Burrill, and Arthur were noted as well as those of Prillieux in France on the bacterial decay of wheat grains and of Reinke and Berthold in Germany on bacterial rot of potato tubers. That De Bary still regarded bacteria as of minor importance as pathogens of plants is shown in his statement that "saprophytic bacteria may also, under special conditions, attack the tissues of living plants as facultative parasites, produce disease in them

and destroy them. But this only confirms what was said above, that bacteria are not objects of great importance as contagia or diseases affecting plants."

The validity of Smith's work on bacterial diseases was challenged in 1897 by Alfred Fischer, professor of botany in the Königlichen Gymnasium at Leipzig, who had studied with De Bary in 1881. In a book entitled "Lectures on Bacteria" (36) he denied the reliability of the results of Smith and others who claimed to have seen bacteria in plant cells. Smith replied to Fischer in 1899 (75), pointing out his complete lack of sound knowledge of the subject. Fischer replied the same year (37), and Smith replied in two rebuttal papers, which seemed to end the controversy (76, 77). While this debate is now chiefly of historical interest, it is significant that more than 20 years after the work of Koch and of Burrill a rather prominent botanist still challenged the existence of bacterial plant pathogens.

EARLY WORK WITH VIRUS DISEASES

About 1870, there occurred in the tobacco-growing region of Holland a disease of this plant which reached such damaging proportions that farmers sought the help of government scientists. Adolf Mayer, director of the agricultural experiment station at Wageningen, undertook a study of the disease about 1880. He published his report in Dutch in 1885 and in German in 1886 (57). He was German-born and German-trained and was apparently acquainted with the work of Koch. In searching for an explanation of the cause of the disease, he studied soil fertility, air-temperature changes, cultural methods, seed transmission, and nematodes but did not relate any one of those factors to the etiology. He described the disease, and, from the mosaic pattern common on leaves of affected plants, he suggested the name "mosaic," which has been retained until the present as the common name of the disease. Mayer discovered that when he macerated the tissue of a diseased leaf and injected the juice into the midrib of a healthy plant, the latter, after about 10 days, began to show typical symptoms. These appeared, not on the inoculated leaf, but on the youngest leaves of the growing plant, and as new leaves expanded, they were also diseased. This is the first known record of the experimental transmission of a virus disease of plant or animal. However, Mayer, impressed by the ease of transmission, endeavored to find out more about the infective agency. The possibility of an enzyme was considered, but this seemed to him to be an untenable theory since he removed the infectious agent when he filtered the juice through a double layer of filter paper. Enzyme precipitates with weak alcohol were noninfectious. Continuous heat at 60°C. did not alter infectivity of the juice, but several

hours at 80° destroyed it. He applied the techniques of Koch and others in an attempt to isolate a bacterium which could be shown to be the causal agent. All bacteria isolated were noninfectious, and cultures of a number of known bacteria, when used as inoculum, gave negative results. Mayer closed the investigation, still believing that a bacterium which had escaped him would probably be found to be the infective agent.

About the same time that Mayer was conducting his studies of tobacco mosaic, Erwin F. Smith undertook an investigation of the peach-yellows disease, which had long been destructive in the United States. He was unable to transmit the inciting entity by transfer of plant juice as Mayer had done. He succeeded in transmitting it only by transplanting a bud from a diseased tree to a healthy tree, whereupon the latter eventually became diseased. He received Mayer's paper before he published his final results. He was of the opinion that the two diseases had much in common in the nature of the causal agent (70, 71).

Dmitrii Ivanowski was a Russian investigator at the Botanical Laboratory of the Academy of Science at St. Petersburg. The mosaic disease had become troublesome in the Crimea, and he undertook work with it about 1890 (46). He confirmed Mayer's results with transmission of the causal entity by means of expressed plant juice. He destroyed the infectivity of the juice with heat. He interpreted the causal entity as a bacterium because of the lack of "fungi and other parasites." He did not find, as Mayer reported, that filtration of the plant juice through a double layer of filter paper removed the causal factor, and he pointed out that Mayer was in error in assuming that this procedure would exclude bacteria from the filtrate. Ivanowski passed the infectious juice through a Chamberland filter candle and found the filtrate to retain infectivity. This fact is of great historical interest because it is the first record of the passage of either a plant or an animal virus through a bacteria-proof filter. This technique was so new that even Ivanowski was skeptical, and he was apparently unaware of the great significance of the result. He thought there was a possibility that the bacteria of the tobacco plant in some way went through the filter and made the filtrate infectious, although when he passed liquids which were favorable media for bacterial growth through the candle, they remained sterile for several months. Another explanation of the results, which seemed plausible to him in light of prevailing opinions, was that a toxin secreted by the infectious bacteria in the sap passed through the filter and made the filtrate infectious. The work of Ivanowski thus added one new fact to those proved by Mayer, namely, that the infectious entity in juices from infected tobacco plants passed through a Chamberland filter.

The next investigator to approach this problem was Martinus Willem

Beijerinck (1851–1931), Dutch bacteriologist, who was a colleague of Mayer at Wageningen in the early eighties and moved to Delft in 1887. He took up a study of tobacco mosaic about 1897 (6). He found the juice of infected plants, when filtered through porcelain, to be sterile but still infectious. He sought anaerobic bacteria in the filtrate without success. A minute amount of filtrate was sufficient to inoculate several plants, and he demonstrated that the contagious entity was increased in some way in the infected plant. He called it a "contagium vivum fluidum" and referred to it subsequently in the paper as a virus. Thus when Beijerinck published his results in 1898, he confirmed the findings of Mayer and of Ivanowski and claimed more emphatically than either of them that the causal entity was not a bacterium or any conceivable corpuscular material. He found that the virus would remain infectious in infected leaves which were dried down and kept in the herbarium for two years. He regarded it, therefore, as capable of retaining its existence outside of the plant. He compared the tobacco virus with that of peach yellows and regarded the latter as a contagium capable of existing only in living plant tissue. In the same category as peach yellows he placed the plant variegations of *Abutilon* and other ornamental plants which were transmitted to non-variegated plants by grafting.

Erwin Baur (1875–1933) published a paper on infectious variegation in *Abutilon* in 1904 (5). Baur was a German botanist who later became prominent as a geneticist. He added little to the facts already known but confirmed the fact that *Abutilon* variegation was due to a virus more like that of peach yellows than that of tobacco mosaic. He maintained that the virus could not be an organism, since if a parasite had to depend upon man to make artificial grafts to perpetuate it, it would have long ago disappeared in nature. His argument is not sound in the light of present-day knowledge of insect transmission of most viruses. At that time no one seems to have considered the possibility of insect transmission of plant viruses.

The question as to whether the plant virsues were living or nonliving entities was debated for another 30 years. The events of those three decades will be discussed in the chapter on Virus Diseases. Suffice it to say here that at the beginning of the twentieth century the field of virus diseases of plants was just opening. Two major types that we recognize today had been studied, *i.e.*, tobacco mosaic and peach yellows. It was shown that the causal entities were transmissible and that they increased in the host. One was filterable through bacteria-proof filters. One was capable of existing for a time outside the living host; the other was not. Most of the present store of knowledge about plant viruses was still to be learned.

GROWTH OF PLANT PATHOLOGY IN THE UNITED STATES

Agriculture in the United States, for 200 years after the establishment of the Colonies, was concentrated along the Atlantic seaboard east of the Appalachian Mountains. In the period from about 1830 to 1850 the opening of public lands in the Middle West was occasion for rapid expansion of population and development of agriculture in the newly settled areas. Potato late blight appeared in the United States in the late thirties and was rampant throughout the forties at the same time it was destructive in Europe. As wheat growing expanded in the Middle West, rusts and smuts followed the crop. An indication of the fact that plant diseases were matters of public interest is given by the remarks of Abraham Lincoln in 1859, when, as a former Member of Congress from Illinois, he addressed the Wisconsin Agricultural Society at Milwaukee, Wisconsin, as follows:

> Every blade of grass is a study; and to produce two where there was but one, is both a profit and a pleasure. And not grass alone, but soils, seeds and seasons; hedges, ditches and fences; draining, droughts, and irrigation; plowing, hoeing and harrowing; reaping, mowing and threshing;—saving crops, pests of crops; diseases of crops, and what will prevent or cure them; . . . the thousand things of which these are specimens, each a world of study within itself.

Schweinitz (69) collected and named many parasitic fungi in eastern United States from about 1820 to 1835. He was followed by Ellis and by Curtis. In 1875 Farlow returned from Europe with the stimulation of association with De Bary and at Harvard took early leadership in the study of parasitic fungi.

During the Lincoln administration, Congress passed the Morrill Act (1862), which provided land grants to each state, the proceeds of which were to establish colleges of agriculture and mechanic arts. In the Federal administration, matters pertaining to agriculture were first handled by the Commissioner of Patents. A Department of Agriculture was established in 1862, and it was first represented in the Cabinet by a Secretary in 1888.

Within the Department of Agriculture a Division of Botany was organized in 1885 under F. Lamson-Scribner. Within this division a Section of Mycology was formed in 1886, the name being changed to the Section of Vegetable Pathology in 1887. B. T. Galloway was head of the Section from 1888 to 1890. His early associates were Erwin F. Smith, Miss E. A. Southworth, and David G. Fairchild. Scribner, Galloway, and Fairchild carried out some of the earliest trials with the newly discovered Bordeaux mixture in the United States, especially on grapes, for the control of black rot.

As the section grew, it was elevated to a Division of Vegetable Pathology in 1891, and to its staff were added A. F. Woods, N. B. Pierce, M. B. Waite, W. T. Swingle, J. F. James, P. H. Dorsett, H. J. Webber, Theodor Holm, and M. A. Carleton. As the work of the division was broadened, its name was changed in 1895 to the Division of Vegetable Physiology and Pathology. In 1901 the Bureau of Plant Industry was organized.

In the period from 1885 to 1900 plant pathologists in the U.S. Department of Agriculture carried out numerous pioneer studies. Among these were investigations of fungicides and studies on grape diseases both in eastern United States by Scribner, Galloway, and Fairchild and in California by N. B. Pierce. Smith studied peach yellows and rosette and later inaugurated his life work on bacterial diseases of plants. Webber and Swingle began studies of citrus diseases in Florida. Smith opened up the work on vascular *Fusarium* diseases of cotton, cowpea, and watermelon. Woods worked on nonparasitic diseases and on tobacco mosaic.

In 1887 Congress passed the Hatch Act, which made an annual appropriation of $15,000 to each state to found and support an agricultural experiment station. This support of agricultural research resulted in the appointment of a station botanist in many of the new experiment stations. Many of these men initiated programs of research on plant diseases in their respective states. Beginning about 1890, plant-disease research began in numerous centers in addition to that started in 1885 in the U.S. Department of Agriculture. Among the early state leaders in the field about 1890 were W. A. Kellerman, Kansas; J. C. Arthur, Indiana; H. L. Bolley, North Dakota; L. H. Pammel, Iowa; B. D. Halsted, New Jersey; G. F. Atkinson, Alabama; F. C. Stewart, New York; F. D. Chester, Delaware; A. D. Selby, Ohio; L. R. Jones, Vermont; R. T. Thaxter, Connecticut; G. E. Stone, Massachusetts.

STATUS OF PLANT PATHOLOGY IN 1900 AND TRENDS IN THE TWENTIETH CENTURY

A hundred years after Prévost, at the opening of the twentieth century, we find a new science of plant pathology well established in Europe and the United States. During the first half of the nineteenth century both animal and plant pathology struggled to define and gain recognition for the germ theory of disease. The lack of adequate techniques to study the life cycles of fungi and bacteria greatly impeded the advance of knowledge of these lower organisms as pathogens. To Brefeld and to Robert Koch goes the major credit of developing methods of pure culture of fungi and of bacteria, respectively. The last quarter of the century saw rapid adoption of these new tools and a corresponding increase in volume of new information on plant diseases.

The last half of the nineteenth century was predominantly a descriptive era in which many new species of pathogenic fungi and a few of pathogenic bacteria were described. Emphasis was laid upon the naming and describing of causal organisms, and correspondingly less attention was given to other phases of disease. Consideration of the influence of environment on disease, which was usually regarded as the sole cause of disease before the De Bary-Kühn period, waned decidedly in this era. Control measures received impetus with the discovery of Bordeaux mixture, and during the last decade of the century growing attention was given to new fungicides for spraying of foliage and for treatment of seeds. The development of control measures is considered in Chaps. 15, 16, and 17.

By 1900, bacteria as plant pathogens had been finally accepted. Virus diseases had been recognized as a distinct type of infectious malady, but most knowledge concerning them was still to be attained.

In the twentieth century, phytopathology has expanded so far and so rapidly that we can undertake to mention here only the major lines of development. This period will, in the future, be distinguished by a broadening of plant pathology into a well-rounded science which in its interrelation with other fields embraces the study of plant disease in its broadest aspects. There may be mentioned several of the major lines of emphasis.

1. The relation of environment to disease development; its influence upon the host, upon the parasite, and upon the interaction of host and parasite.
2. The nature and inheritance of variability in the pathogen with particular reference to its pathogenic property.
3. Virus diseases; the chemical nature and the biological activity of plant-infecting viruses.
4. The nature and inheritance of host resistance to pathogenic organisms; development of disease-resistant varieties of crop plants.
5. The improvement of fungicides and of methods for estimating their fungicidal and fungistatic properties.
6. The prevention of disease through regulation, quarantine, inspection, certification, and education.

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CHAPTER 3

NONPARASITIC DISEASES

We have pointed out in the previous chapter that before the acceptance of the germ theory, diseases were usually attributed to the effect of various adverse climatic factors. Throughout the latter half of the nineteenth century a majority of the research in plant pathology was directed toward description of plant pathogens. The field of pathology concerned with those diseases in which no infectious agency was involved received relatively little attention. Sorauer was one who continued to emphasize the importance of the nonparasitic diseases. In the third edition of his text (1909) he devoted a full volume to this group. An English translation of this volume by Miss Frances Dorrance was published in 1922 (9).

One way of approach to an analysis of the various causal factors which enter into disease development is through a consideration of those maladies in which no parasitic agency is concerned. A disease, defined in the broadest sense, is any departure from the normal in structure or in function. Obviously deleterious changes in the external or internal environment of plants may bring about permanent effects and produce symptoms which justify their designation as those of disease. In Chap. 13 it is pointed out that in the study of the effect of environment upon the development of parasitic disease one needs to consider the effect of the environment upon the host, as well as upon the parasite and upon the interaction of the latter with the host. Much loss of economic crop plants is brought about through the effect of adverse environment without the intervention of a parasite. Certain of the better-known types of nonparasitic disease will be described.

LOW-TEMPERATURE EFFECTS

It is well recognized that certain species of plants or, indeed, varieties within a species have more favorable conditions for growth in a warm climate; others are characteristically cool-weather forms. Some are notably resistant to low temperatures and frosty weather; others are very sensitive. Corn is generally considered a warm-weather plant; cabbage, a cool-weather one. Vine crops such as cucumber and watermelon are very sensitive to freezing temperature; cauliflower, cabbage, and kale are notably tolerant of such exposure. Most species in temperate zones are subject at one time or another to low temperatures and thus to freezing injury.

Injuries at temperatures slightly above freezing are important non-

parasitic maladies. Certain strains of corn are injured by low but nonfreezing temperatures (2). In some varieties of potato, discoloration of the internal tissues of the tuber occurs when the storage temperature is between 0 and 5°C. (1). Necrotic discolored areas appear in uncured sweet potatoes stored at the same range of temperature.

Before describing the effects of freezing injury on plants, it is well to consider what events commonly take place within the tissue when the temperature falls below the freezing point. In tissues of plants ordinarily injured by freezing, the water in the intercellular spaces exists as a vapor or as a liquid film on the exterior of the cell wall. The intercellular water is quite pure, and the freezing point, therefore, is close to 0°C. Within the cell, water present in the central vacuole contains solutes, which tend to depress the freezing point somewhat below 0°. Furthermore, as the temperature of water lowers below 0°, without mechanical disturbance, it commonly undercools several degrees without formation of crystals. Inoculation of an undercooled liquid with a single crystal results in complete crystallization of the entire mass. As the temperature of plant tissue lowers, then, a certain amount of undercooling may be expected. When ice crystals form, they do so first in the water of the intercellular spaces. The vapor pressure within the cell becomes higher than that in the intercellular space, and water continues to diffuse outward and to add to the crystalline mass in the intercellular space. The cell tends to shrink, and the freezing point of its contents lowers. The first process, then, is the formation of intercellular ice without damage to the cell contents, a process which may proceed while the temperature drops several degrees below 0°. Rise of temperature above 0° results in dissolution of crystals in the intercellulars, and diffusion of water into the cell may proceed without perceptible injury. If the freezing process advances to the point where ice crystals form within the cell, injury to the protoplast follows, and usually the plasma membrane is broken and the cell dies. Characteristic of plant tissue is the fact that cells and tissues differ in freezing point and in sensitivity, and therefore differential injury is common. In some plant tissues ice fails to form regardless of the temperature. This is the case with those hardy organs which commonly withstand freezing weather. In some plants repeated exposure to temperature at or just below freezing results in a conditioning of the cell contents so that they withstand lower temperatures than when not so treated. This is commonly referred to as "hardening." Some plants such as cabbage, for instance, "harden" readily, while others, tomato, for example, do not.

The protoplasm of the cells exposed to freezing temperatures is subject to various types of injury. The process described above results in gradual

dehydration of the protoplasm, and at some point this results in salting out or coagulation. Although the cell may not die, the protoplast may not return to its original condition. Upon thawing, the diffusion of water into the cell may result in injury from stretching of the protoplast until rupture of the ectoplasm and the plasma membrane occurs.

Intracellular ice formation depends upon whether the freezing point of the cell sap can drop (and thus become undercooled) as rapidly as the temperature drops. Speed of cooling is therefore important as well as the speed of exosmosis of water and consequent reduction in freezing point. Rapid freezing is more injurious than slow freezing since it is more likely to induce intracellular ice formation. The speed of diffusion of water through protoplasm to centers of extracellular ice formation varies with plants (and with tissues within the plant). In tender plants cellular permeability to water is very low. Exosmosis becomes less rapid as temperature decreases, and thus when ice starts to form in undercooled tissue, the opportunity for water diffusion is much less. Excessively undercooled tissue is much more dangerously predisposed to freezing injury than normal tissue.

Thawing of undercooled tissue or that containing extracellular ice has dangers. Ice thawing fills intercellular spaces with water, which rapidly penetrates the cell wall, and the latter expands. If the protoplast does not absorb the water quickly enough, a layer of water may accumulate between wall and protoplast, and a consequent tearing or other injury to the latter may follow. Hardy plants are injured less than tender plants, in part because their protoplasts can expand as rapidly as their walls. After thawing is complete, the protoplast, though alive, may be more sensitive to such factors as high turgidity. It may be less reactive to moisture changes and thus to repeated freezing and thawing.

In general, then, it may be said that freezing injury to plants is the result of extracellular or intracellular ice formation or both. The pathological symptoms which result are based upon the differential sensitivity of various cells and tissues to freezing temperatures. When cells or groups of cells die, they usually discolor, and their location is evident as brownish areas in the tissue. When this injury occurs in actively growing plants or tissues, the resulting clinical picture varies. Abnormal cell division, callus formation, and other responses characteristic of wounded tissue may follow. Hormone activity may also be affected with consequent stimulation of otherwise dormant growing points.

Three important types of freezing injury will be considered here: (*a*) injury to dormant storage organs such as potato tubers; (*b*) injury to perennial plants, which are exposed to freezing and thawing of fall, winter, and spring months; (*c*) injury to succulent herbaceous plants exposed to frosty weather during the growing season, as in the case of injury to garden pea.

Freezing Injury of Potato. When potato tubers are kept for a long period at 0 to 5°, they take on a sweetened taste. This is not associated with freezing but occurs at temperatures above freezing because of the fact that starch is converted to sugar in the tuber tissue more rapidly than the latter is used in the normal slow respiration which proceeds at that temperature. Potatoes in this condition are not suitable for culinary use, especially frying, since an undue amount of caramelization of sugar causes an undesirable color. Sweetening can be readily corrected by storing potatoes so affected at 5 to 10° for a few days before using. In some varieties stored for a long period just above freezing, an internal breakdown of the tissue known as mahogany browning occurs (1).

Freezing or frost necrosis is the condition which results when the temperatures to which tubers are exposed are such as to cause differential effects in the tissue. The greatest danger of this injury is in the autumn in northern states, when early frosts often come before or during the period of harvest and removal to storage or market. The symptoms of freezing necrosis have certain characteristics which help to distinguish them from those of other tuber diseases. A knowledge of the history of a given lot of potatoes is desirable, however, in the completion of a diagnosis, since freezing-injury symptoms may overlap those of other tuber diseases (4, 12, 13).

There is seldom any external sign of injury. The tissues at the stem end are in general more sensitive to freezing injury than those of the eye end, and the differentiated vascular cells such as tracheae, sieve tubes, and tracheids are more susceptible than parenchymatous cells. Three types of necrosis are common, and a given tuber may show one or more of these (Fig. 1). The first type, known as ring necrosis, consists of discoloration, particularly in the region of the vascular ring and more commonly at the stem end. Net necrosis consists of blackening of the finer vascular elements of the inner phloem, which are scattered throughout much of the flesh of the tuber within the vascular ring. The blotch type is less easily defined but usually comprises irregular areas of various sizes ranging in color from an opaque gray to a black. In general, these three types appear in the order named as the duration of the freezing period is lengthened. They may therefore be expected to overlap. Another common occurrence is that individual tubers in a given lot differ in the point to which they undercool before ice formation occurs, although the actual freezing point (the temperature to which the tissue rises immediately after undercooling) of each may be similar. This also leads to variation in necrotic pattern. In general, potatoes stored at 0°C. or slightly above for several weeks have a freezing point about one degree below those stored at 10°. When the drop in temperature is rapid, the degree of under-

cooling before ice formation and freezing is ordinarily less than when the reduction in temperature is more gradual. All of the factors influence the extent of freezing injury and the type of symptoms which follow.

Freezing Injury to Alfalfa. Winter injury to stands of biennial or perennial legume forage crops is often a destructive malady. Of these, alfalfa, being a perennial crop, suffers most in the North Temperate Zone. All winter injury is not necessarily freezing injury since some of it, at least, results from suboxidation when ice sheets occur. We shall be concerned here with the effect of freezing on alfalfa roots and crowns, which has been studied in detail by Jones (3).

FIG. 1. Freezing injury of potato. *A*, ring type; *B*, net type; *C*, blotch type. (*After Jones, Miller, and Bailey.*)

When the upper taproot and the crown of stems are examined in cross section, it may be seen that the annual increases in diameter take on characteristics not unlike those of annual rings of woody plants. It is thus possible to relate the regions of injury to the period of growth at which the injury occurred. The parenchyma cells make up a more or less continuous mass from the phloem parenchyma at the exterior by way of the phloem and wood rays to the pith or pith-like cells at the center of the root or stem. In this tissue the greatest damage results. Even when this is severely damaged in first-winter plants (where it occurs characteristically in the outer phloem tissue, resulting in a ring of dead cells around the root), the surviving cambium and vascular bundles may regenerate a new root system to sustain the above-ground buds. In older roots the damage is more often in the phloem and phloem rays of the last season's growth and in the inner phloem parenchyma. The injury is thus embedded well within the root, but it does not so frequently extend to the center of the

root as it does in young plants. There is also a certain amount of injury to xylem bundles.

The injured cells darken in color. Whenever they occur, usually in groups, the adjacent uninjured parenchyma cells become meristematic and begin to lay down cork layers around the injured areas. Thus in time patches of dead tissue may be found walled off in the pith, in the wood, or in the phloem; these are no longer of any consequence in the growth of the plant. It thus becomes possible for a plant with a severely injured root to recover and continue vigorous growth. This is made possible by the differential response of the root tissue to freezing. Cambial regions seem to be the least vulnerable, and they also act most promptly in a regenerative role, while other surviving parenchyma cells adjacent to dead regions wall off the dead tissue, thus protecting the root from damage by facultative parasites. Freezing injury of alfalfa is a common predisposing factor to infection by the bacterial-wilt organism, *Corynebacterium insidiosum* (McCull.) Dows.

Winter Injury to Apple Trees. Low temperatures are the cause of various types of damage to fruit trees. In the apple at least three types of damage occur in northern states. One of these is commonly referred to as winter sunscald, in view of the fact that symptoms develop on the sun-exposed side of the tree. Dead patches of bark appear in the late spring, and as they peel off, the sapwood is exposed in the lesion. Eventually a canker is formed, and as the tree grows in successive seasons, the lesion is surrounded by a callus border formed by the growing portion of the bark and sapwood. Mix (7) concluded that winter sunscald was due to freezing to death of the bark, cambium, and sapwood in late winter by the rapid temperature fall at night following the increase in temperature of the injured region by the sun's rays during the day. Injury to trees in the crotch or at the crown he considered to be distinct. Crown rot appeared to result from early winter injury owing to a lower degree of hardiness in the tissue at the base of the trunk rather than in the tissue farther up on the tree.

Freezing Injury to Garden Pea. When late spring frosts appear, the foliage of herbaceous plants shows various types of freezing injury. Again the condition of the tissue is a determining factor in so far as the type of damage is concerned. Young meristematic growing tips may be killed while more mature leaves of the plant suffer no injury and organs of an intermediate stage of development suffer only in localized spots. The response of garden pea to late spring frosts illustrates well the type of differential injury and growth responses which may occur (10).

When the night temperature lowers for a few hours to the freezing point for some of the tissues at a time when the pea plant is about 6 in. high,

damage occurs as a rule at the growing tip. This is usually partially enclosed by a young expanding compound leaf consisting of four leaflets, two stipules, and a tendril. Damage may be so slight that only this protecting leaf is affected. The second pair of leaflets then often fails to

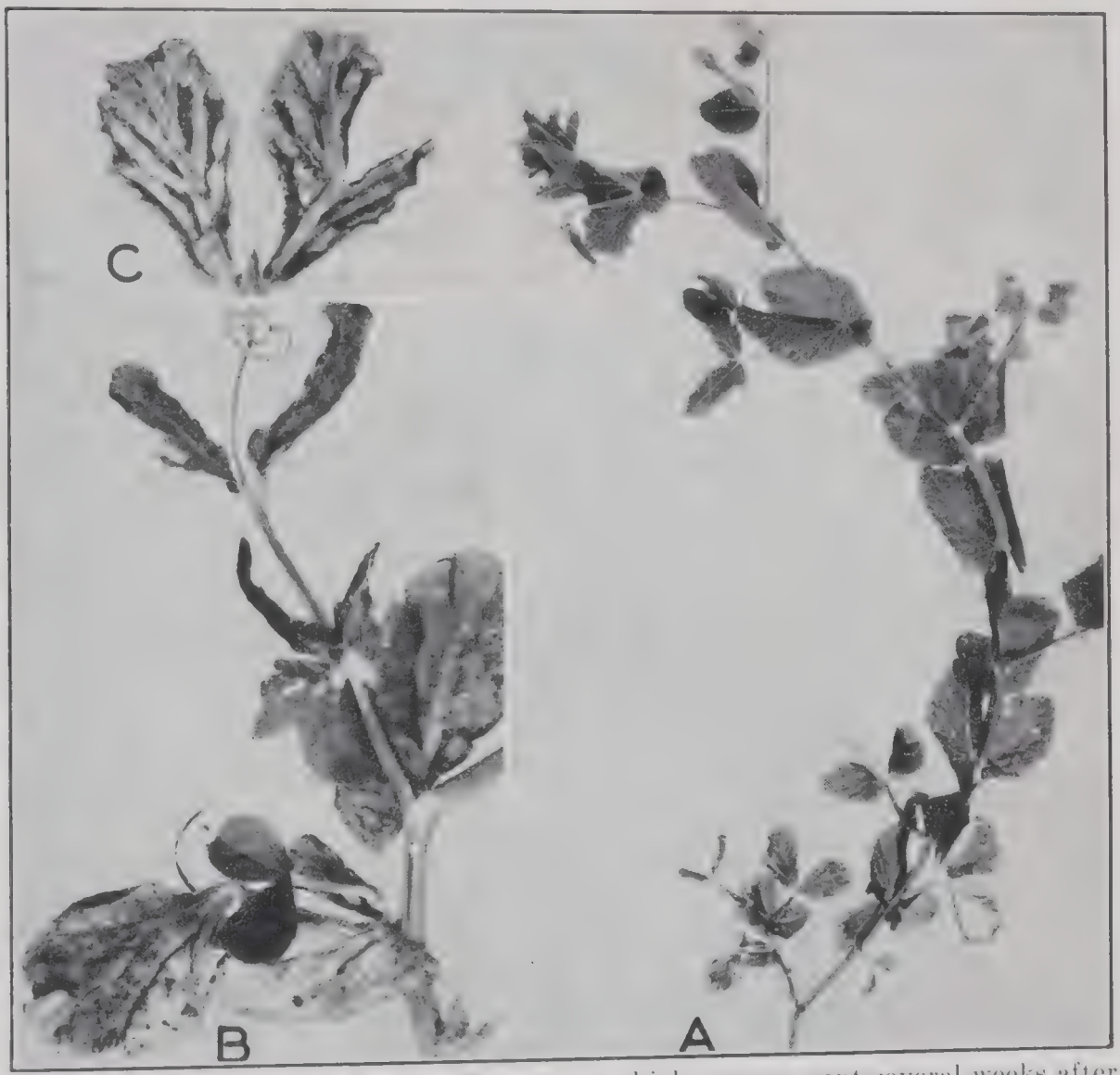

FIG. 2. Freezing injury of pea. Symptoms which are apparent several weeks after injury. *A*, following killing of the growing point at the left, a lower dormant bud grew out to form the main stem at the right; *B*, enlargement of the injured growing point in *A*; in the youngest leaf the stipules and the first pair of leaflets have assumed abnormal shapes, and the second pair of leaflets did not form; *C*, necrotic bands in a pair of leaflets which were developing at the time of injury.

develop while the leaves of the first pair develop in a malformed manner, sometimes becoming bilobed, while the sepals assume an unnatural lanceolate form (Fig. 2). On leaves which are a little older when affected, the leaflets may expand into normal size and shape, and injury is localized in the parenchyma tissue between the main veins. Gradually these areas develop into necrotic bands in which tissue sometimes drops out. Thus when the leaf is a little older, the damage is localized and necrotic, whereas

when it is younger, the damage assumes the form of distorted growth. The young stem below an injured leaf is also commonly injured. The outer cortical cells are so affected that they stop their expansion while the inner parenchyma continues to grow normally. Eventually an elongated rift develops in the outer tissue, and a stem canker results.

When the growing tip is so injured as to preclude further growth, the physiological response of the plant is such that one or more dormant buds at the bases of leaves expand into prompt growth. Presumably this is due to the cessation of auxin formation in the killed growing point, which normally retards growth of axillary buds. The buds near the base of the plant usually are the ones which grow. Commonly, one of them takes precedence, and soon the effect of frost damage is scarcely perceptible. One of the common responses of the plant is that the internodes of the secondary stem expand to greater length than those of surrounding uninjured plants, and before maturity frost-injured plants can be distinguished by the fact that they are taller than healthy ones. When the growing point is not killed, the injured node is recognized only by the malformation of the leaflets at that point.

Thus pea freezing injury may have varied effects. Not only may it cause localized necrosis on various parts of the growing plant, but it may also so modify the plant's physiological processes as to affect subsequent growth distinctly.

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HIGH-TEMPERATURE EFFECTS

Growing plants are commonly subjected to temperatures much above the optimum and sometimes high enough to cause injury. This condition may occur in the soil when subterranean organs are affected or for short midday periods when excessive air temperatures prevail. The effect of high soil temperature in bringing about blackheart of potato through its effect on cell metabolism is discussed below under Oxygen Relations (p. 62). In the case of scalding of tissues in aerial organs it is not easy to distinguish between the effects of high temperature and those of light waves, particularly since periods of extreme heat are usually on clear days when sunlight is bright and more light waves reach the earth's surface.

Heat Injury to Apple Fruits. In occasional seasons a period of several days occurs in which the daily maximum temperature exceeds 38°C. at a time when apple fruits are approaching maturity or are at least more than half grown. Damage to the fruits is sometimes very extensive, resulting in brown or water-soaked tissue beneath the skin, which, as it gradually dries out, leads to depressed areas which are often wrinkled or corrugated on the surface. The symptoms vary somewhat with the variety, and they appear more generally on the exposed than on the shaded side of the fruit.

Many observations show that the temperature of the tissue beneath the exposed surface of a fruit may be much higher than that of the shaded side, and both much higher than that of a shaded apple and the surrounding air. High temperatures do not always result in injury, which indicates that there are other influential factors. Among those which are more obvious are the conditions of previous exposure. Tissue of apples which are exposed to gradually increasing daily maxima will usually stand greater extremes without injury. Limited observations show that the exposed tissue builds up a higher sap concentration. Drought is also a commonly accompanying condition and may be an influential factor. In fact, the greatest damage is recorded when extreme heat suddenly follows an unusually cool period or when it follows a protracted dry spell (1).

Water Core of Apple. Water core is a disease which is aggravated in part by the high temperature of exposed and sunburned apples. It is so called because it is characterized by a watery appearance of the flesh of apple fruits, particularly near the core (Fig. 3). It may start at different points but is always in association with the vascular elements. In severe cases it may involve the entire apple, in which case the watery tissue becomes visible through the skin. It occurs in all apple-growing areas but is most serious in semiarid regions where orchards are irrigated and midday temperatures are relatively high.

For many years it was regarded as due fundamentally to excessive soil moisture and consequent exudation of water into the intercellular spaces of the fruit. This has been disproved by careful irrigation and fertilizer experiments. Heavy irrigation, in fact, usually results in less water core, and fertilization with nitrate or potash fertilizers fails to increase it consistently. Large apples tend to show more water core than small ones, but increasing the size of apples by watering or fertilizing does not necessarily increase water core.

Water core increases as apples become overmature, and picking at the proper time is the best method of prevention. Under some circumstances

FIG. 3. Water core of apple. (*Courtesy of U.S. Department of Agriculture.*)

the disease will be reduced in storage. Neither the type of wrapper nor the type of storage has any consistent effect on the degree of recovery. Some varieties recover more effectively than others, and small-sized apples make a better recovery than large ones. There is a tendency for water-core apples, if they recover in storage, to develop internal browning of the flesh, either localized around the vasculars in some varieties or as a general browning without distinct softening in others. This breakdown appears to be a later phase of water core.

It has become fairly well established that water core occurs most consistently and severely in apples exposed to the sun while still on the tree. Such apples and sunburned ones have a higher sap concentration and a lower acidity than normal apples. Sap concentration increases with degree of maturity. Water-core tissue has a higher sap concentration than normal tissue in the same apple. The disease is apparently brought about by sap

exudation into the intercellular spaces, and it appears that high sap concentration is a precursor of the disease. This condition seems to be aggravated, especially in fruits exposed to the sun, by high air temperatures rather than by the condition of the soil moisture. In fact, heavy irrigation tends to produce fruits with lower sap concentration and less water core. Factors closely associated with carbohydrate metabolism, such as a high leaf-to-fruit ratio, tend to predispose fruits to water core (2-4).

Sunscald of Vegetables. Exposed fruits of tomato, either green or nearing the ripe stage, scald readily during extreme heat. The tissue has a blistered, water-soaked appearance, and rapid desiccation leads to sunken areas, which usually have a white or gray color in green fruits or a yellowish cast in red fruits. Varieties in which heavy foliage is characteristic and in which there is greater protection from the sun's rays usually have least damage. A similar type of injury to onion bulbs and potato tubers occurs when they are harvested in bright hot weather. Rose and Schomer (14) exposed potato tubers to infrared rays and found the tissue at the surface to be 9 to 23° higher than the air immediately adjacent to it. Sunscald was regarded by them and by Nielsen (11) as a major predisposing factor to bacterial soft rot (p. 101).

Succulent foliage of many plants suffers from sunscald. Potato leaflets are among the most commonly affected. The damage appears generally when a period of cloudy, rainy weather promoting rapid top growth is followed by hot sunny days. The scalded tissue appears slightly yellower than the normal green tissue, and the injured region occupies areas irregular in size and shape. This tissue quickly shows lack of turgidity and desiccation follows rapidly, resulting in brownish crisp spots. Sunscald appears and passes through these changes very rapidly and can best be distinguished at the early stages from burning and necrosis of leaves due to other causes.

Heat Canker of Flax and Other Plants. On the black soils of the Red River valley of North Dakota and Minnesota, flax suffers from the effects of the high soil temperatures, which sometimes run as high as 50°C. (13). The injury occurs at the ground level. Young seedlings may break over and die owing to the rapid killing of the succulent cortex. More mature plants show cankers at the soil line, with enlarged callus zones above the lesions, influenced perhaps by the interrupted downward flow of carbohydrates. These weakened plants may eventually break and die if whipped by wind action. Heat canker is most common in widely spaced rows on clean ground where the sun's rays have greatest opportunity to raise the soil temperature. Closely crowded plants or those surrounded by weeds show little damage. The disease may be corrected experimentally by shading the plants or by covering the black soil with

light-colored sand, or by keeping a dust mulch on the surface of the soil. This disease of flax has been reported in Ireland (6).

Similar injury to other crops brought about by excessive soil temperatures have been recorded on beans in Colorado by MacMillan and Byars (7); on conifers, rye, and cowpeas in Nebraska by Hartley (5); on maple, vetch, and peas by Münsch in Germany (8–10); on young plants in southwestern Africa by Schuster (15); and on oak seedlings on the steppes of Russia by Ramann (12).

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LIGHT EFFECTS

There are three conditions of light radiation which result in abnormal or diseased conditions in plants. First of these is reduced light intensity; second, light quality; third, photoperiodic variation. The last of these will not be considered in detail since it seldom results in acute disease development. The fact that the relative length of day and night has a profound effect on the development of various varieties and species of

plants, however, has a possible bearing on their reaction to other enviroral influences and to pathogenic organisms. In a similar way reduced light intensity is important in pathology primarily when it results in plants having a greater predisposition to parasitic organisms.

Etiolation. Etiolation commonly occurs in plants growing under glass in greenhouses, cold frames, or hotbeds. Reduced light from cloudy weather and crowding of plants tend to promote succulent growth, with stem internodes longer than usual, and to retard chlorophyll formation so that the color of foliage is not the normal green. In such spindly plants internal tissue building is abnormal. A predominance of parenchymatous tissue prevails, and the limitation of lignified, suberized cell walls and, in fact, of thicker cellulose membranes leads not only to weak physical structure but to a lack of normal resistance to invasion by parasitic organisms which are ordinarily very weakly pathogenic to stronger plants.

The common disease under such conditions of growth is damping-off. This is brought about by a number of organisms which invade weak plant tissue near the soil line and by rapid ingression bring on a prompt invasion of the parenchyma and a sudden collapse of the plant.

Lodging of grains is influenced by etiolation following too thick seeding or excessive vegetative growth stimulated by excessive nitrogenous fertilization.

Sunscald of Bean Pods. It has been implied that part of the injury associated with sunscald of leaves and fruits may be the effect of short waves, which reach the earth in greater intensity when the atmosphere is clear. Little experimental study has been made to distinguish between heat and light effects except in the case of sunscald of bean, and the disease is therefore of special interest.

In the region east of the Rocky Mountains in Colorado, near Greeley, beans are grown extensively. The altitude is about 5,000 ft. above sea level, and the summer growing period is relatively low in rainfall, with a large percentage of the daylight hours being clear and bright. Sunscald occurs on bean pods as small water-soaked spots, which rapidly turn brown or reddish in color as the tissue shrinks slightly. The spots are commonly confused with those of common bacterial blight. They appear on the exposed sides of pods and, since no pathogenic organism can be isolated, are regarded as nonparasitic in origin.

High temperatures are commonly coincident with development of sunscald. It has been shown, however, that if pods are shaded with coarse cheesecloth in such a manner as to modify the air temperature only slightly, the breakdown may be entirely prevented. This was interpreted as demonstrating that the interposition of a screen, which will filter out the shorter waves of light known to be the most injurious to plant tissue, will prevent

the disease, and as indicating quite definitely that the basic cause of sunscald of bean was light injury rather than heat injury (1). Further experimental study with infrared and ultraviolet rays showed that only the latter produced typical symptoms (2). It would be of interest to know whether light is influential in other sunscald diseases.

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SOIL-MOISTURE DISTURBANCES

It is natural to expect that plant species vary in their reaction to soil moisture. Some, such as celery, have as an optimum a relatively high moisture level; others are particularly sensitive to excessive moisture. In agricultural practices crop plants are sometimes exposed to one extreme or the other and perhaps even more often to rapid changes all of which may cause distinct pathological effects. As in high- or low-temperature effects the rate of change is often the most influential factor, in that it may determine the extent to which a plant can withstand extremely high or low soil moisture.

Excessive Water Damage

Flooded fields or areas within fields commonly occur after heavy rains when drainage is inadequate to remove water promptly. Flooding injury which follows is common. If water stands for 48 to 72 hr. in midsummer heat, succulent annuals such as potato, cabbage, and turnip usually become permanently wilted. When such plants are pulled, there is usually to be found general decay of the fibrous root system and soft rot of storage organs. Flooding injury is sometimes attributed to the direct effect of reduced oxygen supply upon subterranean organs. The true nature of the cause is much more complex. The disturbed effect of flooding on the soil microflora is a factor. The encouragement of anaerobic organisms probably leads to formation in the soil solution of toxic materials, such as nitrites, which have a poisonous effect upon higher plants. Once serious damage is suffered, facultative parasites, otherwise noninjurious, serve to hasten the breakdown.

Irregular Water Supply

Some specific diseases result from irregularity in water supply rather than from high or low moisture levels. This group of diseases is in general poorly understood. They include lettuce tipburn, blossom-end rot of tomato, and blackheart of celery. The sequence of events which lead up to the development of the last two of these has been studied critically.

Blossom-end Rot of Tomato. This is a very common and destructive disease of outdoor and greenhouse tomatoes. It is characterized by a lesion at the blossom end of the fruit, which occurs while the latter is still green or while it is ripening on the vine. A water-soaked spot appears at the point of attachment of the senescent petals. It enlarges quite rapidly until it is an inch or more in diameter, and at the same time the diseased tissue shrinks, depressing the surface of the lesion. The color becomes darker until eventually it appears nearly black. Secondary organisms commonly invade the tissue. Ordinarily the diseased portion

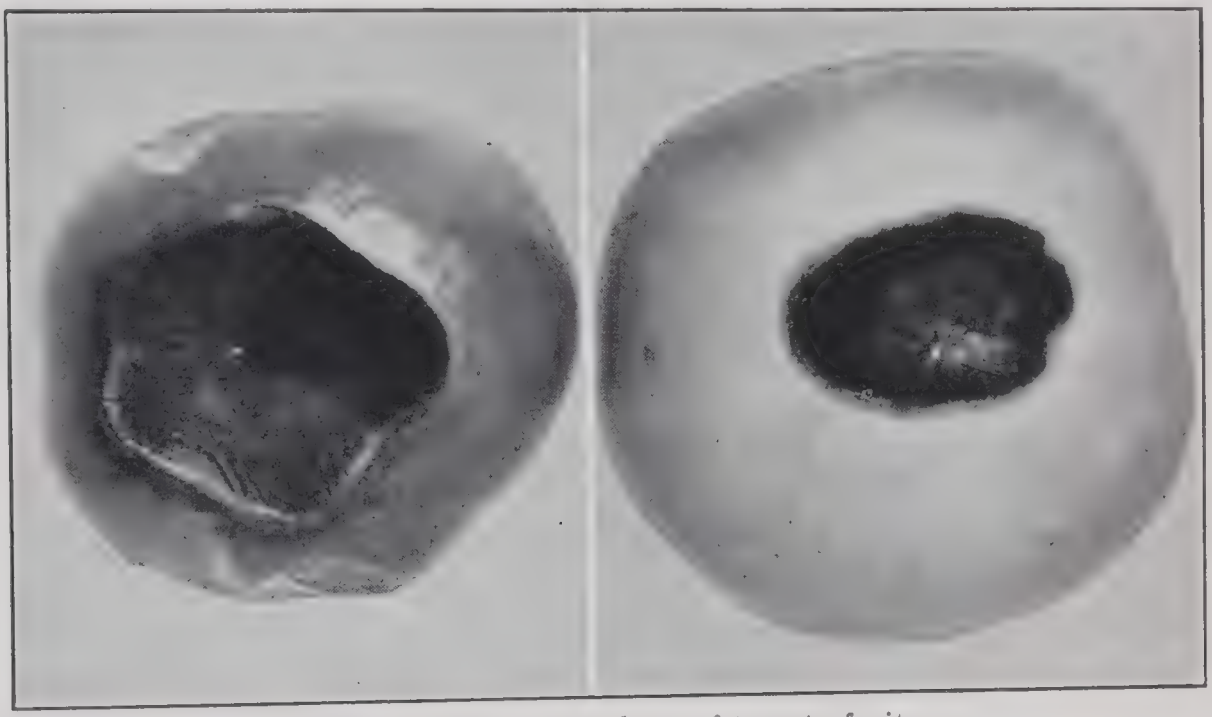

FIG. 4. Blossom-end rot of tomato fruits.

of the fruit becomes delimited as a sunken, leathery, dark-colored area (Fig. 4).

If tomatoes are grown in soil at a relatively constant low moisture level, they become adjusted to such environment, and although they do not grow thriftily, they ordinarily do not develop blossom-end rot. If plants are grown with adequate soil mositure to produce rapid succulent growth and to promote a high rate of transpiration, they are more subject to the disease. The course of events which brings it on usually includes a relatively sudden change in the rate of transpiration. This is often the result of reduced soil moisture or irregular fluctuation in the moisture level. Light sandy soils are inclined to fluctuate more widely in moisture and are conducive to blossom-end rot. Some fruits on the plant during such a period may succumb while subsequent fruits which develop may mature normally.

Aside from the irregularity of moisture supply at the time of luxuriant

growth, there is little agreement as to the factors in nutrition of the host which contribute to the disease. Foster (3) found that increasing amounts of nitrogen were apparently conducive to the occurrence of the disease when other conditions favored it, while it was reduced with increasing amounts of phosphorus. Robbins (8) studied the effects of nutrient solutions of different concentrations. At the lowest concentration, where growth was greatest, no disease occurred, while at the highest concentrations, where there was widest fluctuation in rate of transpiration, 80 per cent of the fruits became diseased. That transpiration has an influence on blossom-end rot is indicated by the work of Schroeder (9), who reduced the disease by spraying with a substance which lowered the rate of water loss. Eaton (1) in a study of the toxicity of chloride and sulfate salts in nutrient solutions found blossom-end rot to increase with increase in concentration of either type of salt. The accumulation of calcium and magnesium was thought to be an important contributing factor. Raleigh and Chucka (6) found the disease to be induced when the nutrient solution was relatively high in nitrogen, sulfur, magnesium, potassium, or chlorine and when it was low in calcium. Whenever the calcium content of the fruit fell below 0.20 per cent, the disease occurred on some fruits. The balance of nutrient elements was of more importance than the concentration. In a comprehensive study of 87 nutrient solutions, which varied in the balance of calcium, magnesium, and potassium cations and in nitrate, phosphate, and sulfate anions, Lyon *et al.* (5) found that blossom-end rot increased as calcium decreased.

It is commonly observed that varieties of tomato differ in their susceptibility to blossom-end rot (10), but variety reaction differs widely. Inasmuch as the disease is the result of a transient physiological abnormality and since varieties differ widely in their rates of fruit set and maturation, a given variety may be very susceptible to the disease when the environment happens to favor it in one situation and may be in a more resistant state at the critical time in another situation.

Blackheart of Celery. The blackheart disease is almost coexistent with celery culture. It appears usually after midseason and often develops suddenly. Sometimes an entire field becomes unmarketable because of this malady.

The first symptoms usually appear on the young leaves although the outer older leaves may become chlorotic first. The heart leaves shrivel and turn dark. The necrosis progresses rapidly to the petioles of the young leaves, and soon the entire heart becomes black, surrounded by the unaffected petioles of the outer leaves. The observational evidence in many states offers little in the way of uniform interpretation of the causal nature of the disease (2). In many cases it is found to develop rapidly after a

protracted dry spell followed by a heavy rain. Flooding alone does not appear to be sufficient to bring it about (7).

The best evidence that irregular water levels in the soil are a main feature of the environment inducing blackheart was furnished by experiments in Florida (4). In the intensive celery-growing area near Sanford the crop is grown on subirrigated land, where the water content of the soil can be regulated since rain is infrequent during the main part of the growing season. When celery is grown at a relatively constant low moisture level or high moisture level, blackheart is not induced. When the soil in which optimum moisture has been maintained is allowed to drop to a low moisture level for a protracted period and the moisture level is then raised suddenly to a high level, blackheart usually follows quickly and severely. In this area the disease is successfully controlled by maintenance of a fairly uniform optimum moisture level. The condition in the plant which seems to favor blackheart, then, is a slowing down of growth by lowering the moisture supply followed by a sudden return to a level above the optimum. Of many fertilizer combinations tried none was shown to have any effect on the appearance of blackheart.

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OXYGEN RELATIONS

Blackheart of Potato. The conditions under which this disease develops vary. One environment is that provided formerly in shipment of

potatoes in boxcars in midwinter from northern points in the United States. With poorly insulated cars it was customary to provide heat from a wood stove, which could be fired by the attendant if and when freezing weather was encountered in transit. The heater placed in the center of the car of course provided uneven temperatures. Often potatoes in sacks near the stoves were cooked, while those at the ends were frosted or frozen. In between these extremes the potatoes tended to show blackened centers when sliced open, or there soon developed following slicing a fairly rapid change in color from white to pink to brown to dark brown or black (Fig. 5). This trouble, known as blackheart, was also found in poorly ventilated storage bins, particularly in tubers at the bottom of deep piles. It has also been found occasionally in tubers in the field, particularly in those

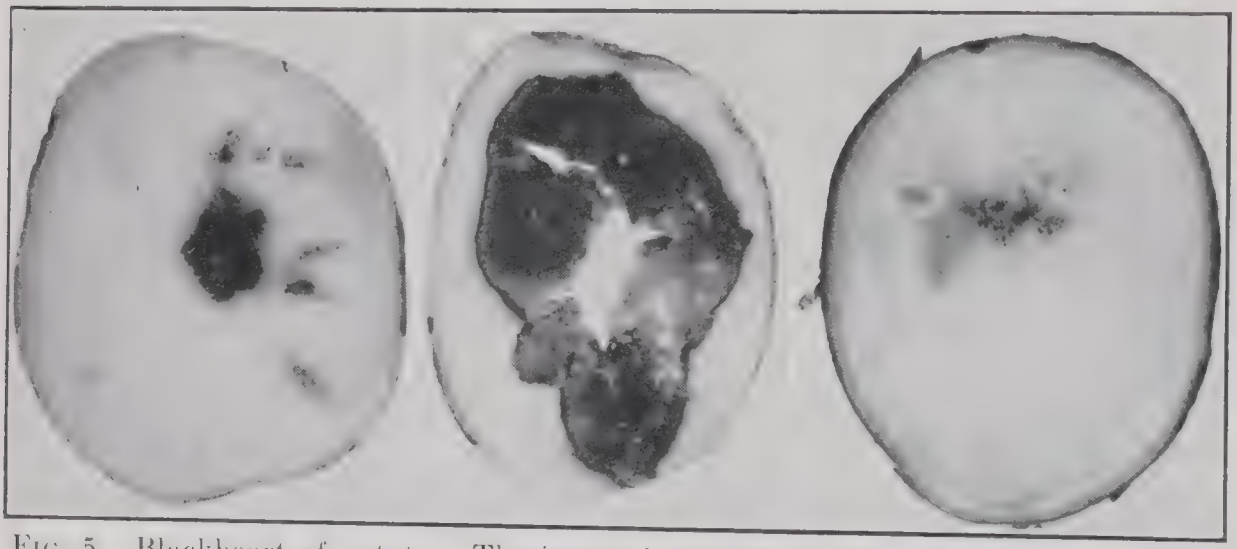

FIG. 5. Blackheart of potato. The internal necrosis in the specimen at the right developed while the tuber was maturing in warm sandy soil.

areas where the temperature rises to a high point during periods of tuber growth and maturation. Thus it is clear that a number of rather distinct sets of environments produce practically the same result or cause the same disease, *i.e.*, high temperature in transit, poor ventilation in storage, high soil temperature during the growing season.

The fundamental nature of blackheart was revealed by the work of Bartholomew (1, 2). He found that he could produce blackheart in tubers by exposing them to high temperatures for 24 to 48 hr. in an oven at 40 to 42°C. Above and below this temperature production of the disease tapered off. No blackening was observed below 38°; little was produced above 50° and none above 60°. At the higher temperatures, of course, actual cooking occurred. At the optimum temperature (42 to 44°), the disease was prevented if a stream of free oxygen was passed through the chamber during exposure. This showed that oxygen was an essential factor and conversely that lack of it contributed to the disease. Mann

and Joshi (6) in India produced blackheart in 3 days at 36° and in 6 to 12 days at 27 to 30°, if they first coated the tubers with collodion or paraffin or replaced the air in the container with a carbon dioxide or nitrogen. Stewart and Mix (9) showed that they could produce blackheart at low temperatures by placing tubers in sealed containers. This pointed also to oxygen as the important limiting element.

In a study of the progress of disease development, Bartholomew showed when a tuber exposed to 40° for 24 hr. is cut open, the interior tissue appears normal at first but, upon exposure to the air, pink discoloration occurs, which gradually becomes dark brown or black. This he showed to be an enzymic effect in which certain of the amino acids, particularly tyrosine, were oxidized by enzymes through a series of intermediate compounds to the deeply colored type of relatively stable and insoluble compounds known as melanins. This reaction is a very common one in nature but of course uncommon under average conditions in potato tubers. It is therefore an abnormal physiological process in which one or another type of environal condition, by bringing about suboxidation, upsets the balance in the cell and permits the enzymic process described above to proceed.

High temperature brings about suboxidation by stimulating respiration under conditions where cells in the interior of the tuber cannot secure an adequate supply of oxygen. This results in death of such cells, but the enzymes are not destroyed, and the upset in cell metabolism permits the oxidation of amino acids to proceed. When the temperature of the tuber is high enough to break down the enzymes also (60°), no discoloration occurs and the tubers are merely "cooked."

Poorly ventilated storage over a protracted period provides conditions for blackheart because the respiration of the cells in the tuber, though proceeding at a very low rate, gradually uses up the available supply of oxygen, and cell degeneration again permits the proteolytic process to bring about discoloration. Bennett and Bartholomew (3) showed that in storage the tuber may take up oxygen faster than it gives off carbon dixoide, and if the tubers are in a sealed chamber, vacuum may result. Blackheart did not appear until some 40 days after the oxygen in the atmosphere was exhausted, which was interpreted as showing that the tuber tissue in a sense stores oxygen temporarily and that when the replenishing of cells is not necessarily rapid, as at low temperatures, cells in one part of the tuber may give up oxygen to others needing it. It is this set of conditioning circumstances which probably accounts for the difference in blackheart symptoms produced in storage as compared with those which occur in overheated chambers. In the latter instance the blackened regions are usually solid masses in the center. In the former they may appear in various patterns throughout the tuber.

Bennett and Bartholomew (3) found some difference in varieties in time required for blackheart to develop. Netted Gem or Russet Burbank required longer than other varieties tested. Differences in permeability of skin were thought to have some influence.

Tuber discoloration sometimes occurs in the soil at or just preceding maturity when soil temperatures are unusually high. This phase has not been fully studied, but it is apparent that under some circumstances a somewhat different picture of symptoms may result. The tuber diseases known as sprain and internal necrosis are produced in warm sandy soils and may be a phase of blackheart. Larson and Albert (5) have studied the latter in sandy soils of central Wisconsin, where they found the disease to be correlated with high soil temperature. They controlled the disease by reducing the temperature through the application of straw mulch on the soil during tuberization of the potato plants. Varieties also differ in the degree to which internal necrosis develops, and again Russet Burbank is one of the least susceptible varieties.

Davis (4) showed that discoloration similar to that of blackheart could be produced with electric current, freezing, immersion in toluene or glycerine, and by certain pathogens.

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EFFECTS OF ATMOSPHERIC IMPURITIES

The influence of injurious gases on plants is varied and extensive. The commoner effects are those which arise from atmospheric contamination by industrial plants. However, equally important are those which occur

in storage as a result of extraneous gases or the volatile by-products of the stored plant tissues themselves.

Apple Scald. This disease has been the cause of heavy losses in storage and particularly after apples have been removed from storage to market or the home. The tissue just beneath the skin turns brown to a depth up to one-half inch. The skin may remain firm, or in severe cases it breaks down and sloughs off readily (Fig. 6). Apple fruits so affected obviously lose much of their market value. Scald occurs particularly on the green side of the apple, less so on yellow surfaces, and least on bright-red surfaces. Thus the disease develops more in fruit which was immature rather than well matured when picked. Fruit is more susceptible when picked from

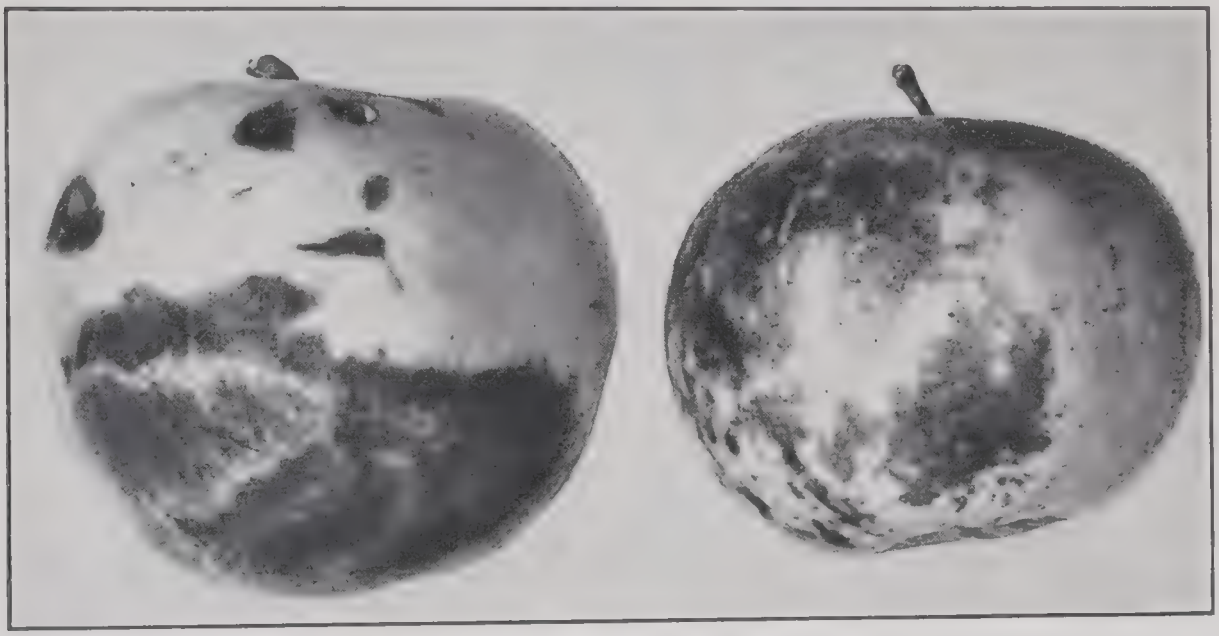

FIG. 6. Apple scald. (*Courtesy of U.S. Department of Agriculture.*)

trees forced by excessive nitrogenous fertilization, heavy irrigation, or high rainfall. In storage, low temperature and good ventilation reduce the amount of scald which develops even after apples are removed for marketing.

It has been proved that one or more of the volatile constituents which are associated with the characteristic odor of apple fruits is the major cause of scald. In ordinary cold storage this material increases in concentration in the atmosphere to a point where the cells of the tissue beneath the skin are killed. Where potatoes are stored in the same room as apples, it has been noted that the volatile materials from the latter delay the normal sprouting of the tubers (10). This same material has been shown to have certain mild fungicidal properties also (5).

The discovery that the injurious volatile material is absorbed by mineral oil has provided a very effective means of control (2, 4, 22). The mineral

oil may be applied in one of two ways. It is introduced into the wrapper when the fruits are packed in individual papers, or it is applied in shredded paper which is mixed in the barrel pack. This practice, together with provision of air changes in the storage and the use of ventilated containers, has served to reduce greatly the damage from the disease. Gerhardt (11) showed that an oilpaper wrap adsorbed about ten times as much volatile material as one of plain paper. Smock and Southwick (19) controlled scald by passing the storage-room air through a canister of activated coconut-shell charcoal, which removes the injurious gases.

Other Gaseous Injuries in Storage and Transit. Discoloration of yellow or red onions in storage is brought about when leakage in the cooling plant permits small amounts of ammonia gas to enter the atmosphere. Outer scales of red onion turn greenish black while those of yellow onions turn yellowish green. This is the result of the absorption of ammonia in particles of moisture present on the surface and the color reaction to the alkaline solution by the flavone and anthocyanin pigments present in the onion scales. Similarly, certain discoloration occurs when apples, peaches, pears, and bananas are exposed to ammonia fumes. In all cases the direct reaction is a color change. When the concentration of ammonia is high, there may be secondary breakdown of the tissues in the form of softening and darkening of the cells (16).

Fumigation of products is sometimes necessary. Ramsey (15) described a case where treatment of watermelons from California with formaldehyde gas was required before they could be shipped into Washington. This was a quarantine requirement because of the foot-and-mouth disease of cattle. The treatment resulted in a severe damage to the melons in the form of blisters and sunken areas, which appeared about 27 hr. after fumigation. Stewart and Gloyer (23) studied the use of formaldehyde gas for the disinfection of seed-potato tubers affected with common scab (p. 147). Injury appeared in the form of sunken brown spots surrounding the lenticels and eyes. The amount of injury decreased roughly in proportion to increase of the amount of potatoes per cubic foot of space.

Illuminating-gas Injuries. Illuminating gas is injurious to plant growth and may cause detrimental effect upon vegetation in several ways. One of these is by the escape of gas from street mains in such small amount as not to be detected by ordinary means but sufficient, when escaping continuously, to injure the roots of shade trees in the vicinity. The result is usually a protracted period of decline in which a variety of symptoms may appear on leaves, branches, and trunk.

The escape of gas into the air from leaks or incomplete combustion is sometimes damaging in residences and greenhouses. Young succulent plants are sensitive to some of the constituents of illuminating gas. Ethyl-

ene is one of the common poisonous impurities. Species of plants vary in their sensitivity. Carnations are one of the most sensitive; 1 p.p.m. may prevent buds from opening properly. Epinasty of leaves and petioles is a common early symptom, and the most sensitive forms, such as tomato, may be used as detectors. Properly controlled, ethylene may be used effectively to retard or hasten blooming and other processes, but when allowed to occur in excess, it brings on pathologic effects in the form of retarded growth, yellowing and falling of leaves, reduced productivity, and general unthriftiness (6–9, 12, 17, 26–29).

Smelter-fume Injury. The damaging effects of gases from smokestacks of industrial plants, particularly those from smelters and chemical factories, have caused heavy losses and received much attention by investigators. The most common damaging constituent of such fumes is sulfur dioxide. This leads to acute bleaching and killing when the concentration is suddenly high but more often to chronic slowly developing symptoms from a low but more or less consistent concentration in the atmosphere. The symptoms consist in a variety of forms from yellowing and reddening of tissues of leaves to defoliation and stunting. Herbaceous annuals and perennials may be stunted, ripen prematurely, and storage of starch or sugar may be reduced. The damage is influenced by environal conditions. Species vary in their sensitivity. Legumes are very susceptible; cereals and grasses moderately so; beets, potato, brassicas, and iris are quite tolerant. The damage is greatest when temperature and humidity are high.

The indefinite nature of this damage and the variety of almost immeasurable and indistinguishable effects have led to much controversy and litigation over smelter-fume damage. In recent years this has been reduced by improved stacks and by greater control of areas in the vicinity of smelters, and by more rigid enforcement of ordinances requiring efficient stoking of coal furnaces in residential areas of cities (1, 13, 14, 24, 25, 30).

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LIGHTNING INJURY

Although the annual damage to plants from lightning is relatively small, it nevertheless is important because it is sometimes confused with other diseases. The nature of damage to large trees is familiar to everyone. Damage to herbaceous crops is more commonly overlooked.

Lightning striking the ground causes the spread of high electric potential in a roughly circular direction. The extent of such spread before it becomes dissipated will depend on the nature of the soil and the vegetation covering it. Commonly rain precedes the discharge, and if it follows a dry spell,

FIG. 7. Lightning injury of potato. The appearance of an area in which plants were killed, as seen several weeks after the injury. (*After Jones and Gilbert.*)

there may be a relatively thin layer of moist soil underlaid by a dryer zone Such a condition favors a wider dispersal of electric energy and thus a greater sphere of injury. Grasses and cereals are apparently quite resistant to damage since few reports of injury to such crops are on record More succulent plants such as potato, tomato, celery, and brassicas are very readily damaged, and so-called "lightning spots" in fields of such crops are common (Fig. 7).

The occurrence of lightning injury is seldom noticed until some weeks after the stroke. It has then become evident by a roughly circular bare spot, in which most or all of plants are completely dead, having been killed rapidly. At the periphery plants show various degrees of retardation in

growth. The nature of such damage suggests the action of a rapidly progressing fungus pathogen, and, to be sure, secondary organisms may often be isolated from cankered plants. The nature of lightning damage has been studied most critically with cabbage, tomato, and potato.

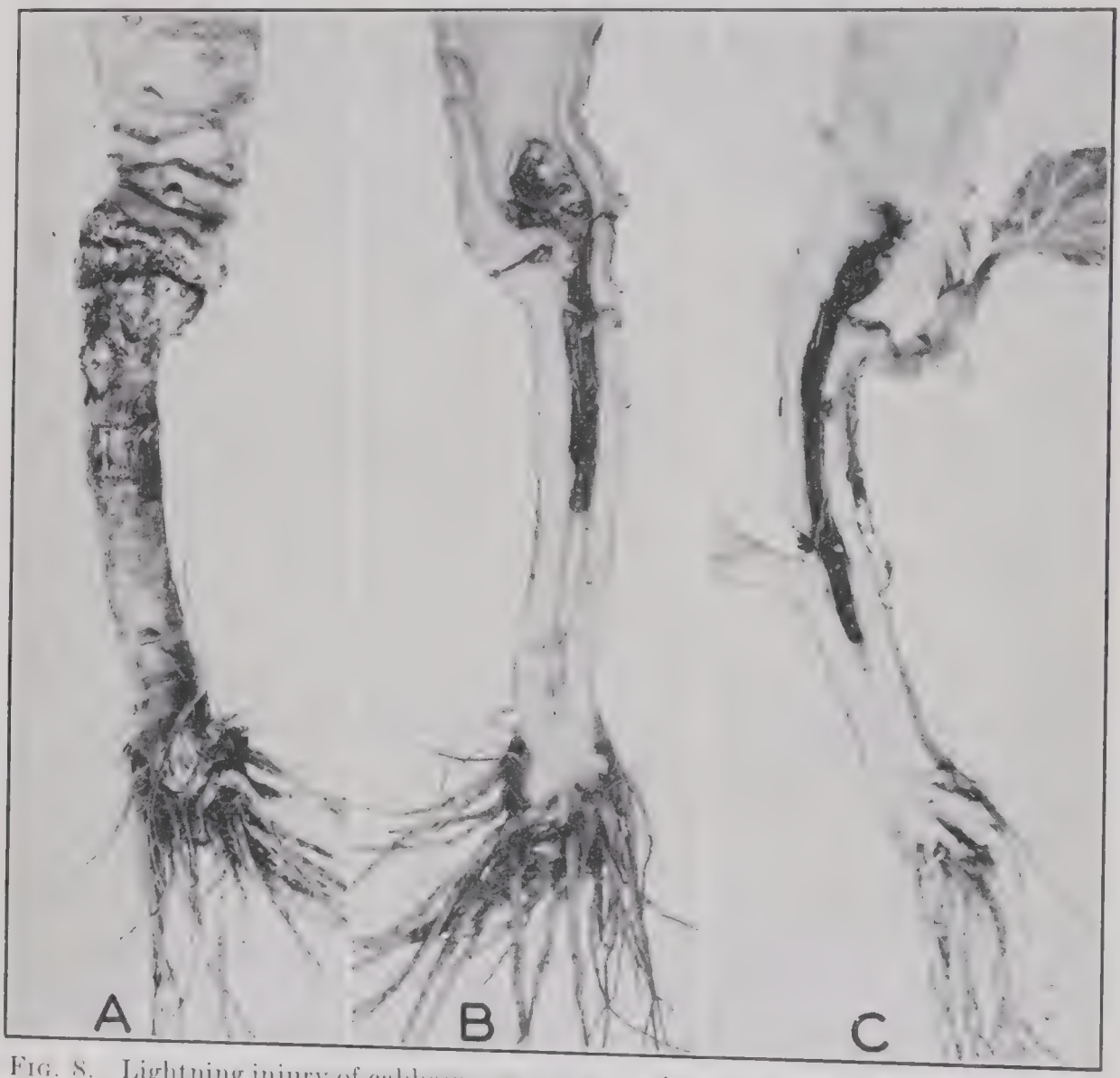

FIG. 8. Lightning injury of cabbage, as seen several weeks after the injury occurred. *A*, callus tissue on the stem at the ground level where the charge entered the plant. *B*, interior of plant shown in *A*. The paths whereby the charge passed through the cortex and the vascular ring are evident. The pith was killed, and as the tissue collapsed, adventitious roots formed in the cavity. *C*, dormant buds stimulated to growth at leaf axes just below where the charge entered.

When the charge is reduced sufficiently to damage the cabbage plant without killing, it penetrates the stem at the soil line with little damage to cortex and vascular ring but permeates the more succulent pith, which is quite extensive in this plant (Fig. 8). The pith cells are killed for some distance above and below the point of entry, and eventually a hollow cavity results, surrounded by a dark brown lining consisting of the desiccated cell

tissue, which has undoubtedly been broken down by enzymic action during the process. This course of events, possibly through the release of certain hormones, stimulates adventitious root development from the inner periphery of the vascular ring, and ultimately innumerable rootlets may almost completely fill the center cavity. The channel of entry through cortex and ring in the meantime is callused over by growth of the uninjured cortical cells. There is a tendency, however, toward abnormal growth stimulation in dormant buds at leaf scars above and below this point. As a rule, adventitious roots are formed at scars immediately above the wound and stem buds at scars below (15).

The tomato is a more succulent plant at midseason than cabbage. The course of lightning effects is the same, but there is greater opportunity for extensive spread of the charge through the pith of the plant. The first symptom appears a few hours after the stroke, *i.e.*, wilting of leaves at the extremities. This is followed by more extensive wilting and withering of leaves and branches and collapse of injured stems due to the damage to and dehydration of the pith. Damage may extend through the pedicels into the fruit, where internal necrosis occurs. This may extend to the surface, where blister-like spots develop which later turn dark brown (3, 17). In potato the pith of the stem is injured with or without killing of the leaves (4, 7, 10, 16). The course of events is not unlike that in tomato. In some cases injury to tubers occurs (12). The effects resemble in some respect those on tomato fruits. Dark depressed areas of various shapes and sizes occur. Sometimes the bud ends of the tubers are shattered by breaks in the skin.

In grape, collapse or necrosis of the pith has been described (1, 2). In cotton (13) and in tea bushes (5) cortical lesions are prominent. In banana the affected pseudo stems have a yellow scalded appearance (11). It is evident that the differential effect of the lightning charge in the plant causes a variety of necroses, and it may upset the physiology of the plant in various ways.

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NUTRITIONAL DISORDERS

It has long been recognized that certain elements are essential for plant growth and normal development. It has also been known that the omission of or pronounced reduction of one or more of the essential elements leads to abnormalities of one sort or another. Likewise an element in excessive amount in the nutrient may bring on unusual appearance and may be pronounced toxic. The types of abnormality are referred to as mineral-excess or mineral-deficiency diseases. The effect of deficiencies on the appearance of plants is often nondescript owing either to the fact that the lack, or excess, of the element or elements concerned causes no acute characteristic symptoms or that symptoms are of insufficient magnitude to remove the development of the plant from that range which it has become the custom to regard as normal. Mineral deficiencies and excesses, however, often cause specific symptoms on different species which may be used as reliable indicators of the cause of the malady.

Essential Elements

The list of essential elements for plants has long included carbon, hydrogen, oxygen, phosphorus, potassium, nitrogen, sulfur, calcium, iron, and magnesium. The first six of these have come to be known as the major elements, while the last four and certain others which have only in the past three decades been regarded as essential (and then in relatively minute proportions), have been commonly referred to as minor elements. The latter group now includes manganese, boron, copper, zinc, and molybdenum. The names "major" and "minor" elements are unfortunate because they imply that all those in the first group are of major importance while those in the second are of minor significance. This is not strictly true since it may be demonstrated that the omission of a so-called minor element may result in an abnormality as acute as or more acute than that resulting from the omission of a major element. The term "trace" element is better applied to those which are required in relatively small amounts as com-

pared with the major elements. It also is generally true that trace elements are toxic to plants when they occur in other than very low concentrations.

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Diseases Due to Materials in Excess

High Acidity. Highly acid soils are toxic to some species. Natural vegetation is thus more or less limited by soil reaction. Among crop plants some are known to be acid-loving, others acid-tolerant, and still others acid-sensitive. When a crop plant is grown beyond its normal acidity range, growth depression commonly follows. There are also various symptoms of disease which occur. They may be largely secondary, however, owing to the fact that an acid soil solution may result in certain elements becoming unavailable or in others becoming soluble to the extent that they are toxic. Examples of this are found in aluminum and manganese salts in acid soils in concentrations which may be very toxic to plants.

High Alkalinity and Mineral Toxicities. Many agricultural soils, in regions of low rainfall, are high in soluble salts. This condition is usually due to the fact that irrigation waters often accumulate salts in the watershed from which they are derived. As the water is applied to crop lands, salts are not leached out as fast as they are applied. Some irrigation waters contain as high as 5 tons of salt per acre-ft. of water. When 2 to 5 acre-ft. per year are used, it may be seen that salts may be applied in tremendous quantities. The salts must be removed in large measure by leaching through the root zone. Soils that drain well are therefore the more desirable.

Black alkali soil has a brown or black crust on the surface caused by a large amount of sodium which is adsorbed on the surface of clay particles. Such soil drains poorly and puddles readily. It can be corrected only by adding calcium salts to replace the sodium and then by leaching out the sodium salts. White alkali, or saline, soils are of better texture and contain chiefly calcium and magnesium salts, which can be reduced by leaching.

The importance of this problem has led to the establishment of the U.S. Salinity Laboratory at Riverside, California.

Naturally, the composition of irrigation waters varies with the watershed from which the river concerned draws its water or with the composition of water in underground reservoirs, when irrigation is supplied from wells. Among the most common toxic ions in southwestern United States irrigation waters are sodium, chlorides, sulfates, carbonates, bicarbonates, and boron. Here again, the effects are in the main chronic and appear as the result of gradual increases in inimical concentrations of one or more ions so that growth depression may progress slowly for a long period without marked symptoms developing.

In all cases plant ranges of tolerance occur, and there are varietal differences within species as well. In the case of boron injury a fairly complete list of relative tolerances has been worked out. Eaton *et al.* (2–4) have classified plants into three groups: sensitive, semitolerant, tolerant. Examples from each group are as follows:

Sensitive		Semitolerant	Tolerant	
Citrus spp.	Persian walnut	Lima bean	Carrot	Alfalfa
Prunus spp.	Black walnut	Tomato	Lettuce	Beet
Grape	Pecan	Cereals	Cabbage	Asparagus
Apple	Common bean	Pea	Turnip	Gladiolus
Pear			Onion	

Boron-toxicity symptoms result from excessive concentration in affected parts of the plant. Boron accumulates more rapidly in some species than in others. The intake of boron is also influenced by the amount of available calcium and potassium. This phase of the problem is discussed later in connection with boron-deficiency diseases. When boron accumulates at the toxic level, chloroplasts are commonly converted to leucoplasts, and the cell contents tend to shrink from the cell walls. Yellowing, browning, and necrosis follow successively, the greatest injury in many crops appearing first at the margins of the leaves. In stone-fruit trees boron accumulates in the bark and in the fruits rather than in the leaves, resulting in dieback of shoots and in brown corky strips on the petioles and midribs.

In humid areas rainfall promotes leaching of soluble salts from the root zone and the excess-salts problems of arid regions do not occur as generally. In sandy soils carbonates gradually leach away, and the reaction of the soil solution often becomes extremely acid. When this condition prevails, toxic concentrations of various minerals may arise.

In extremely acid soil manganese salts become soluble; in alkaline soils they tend to become insoluble. Moreover, plants tolerate only relatively low levels of manganese, although small amounts are essential. Thus

manganese toxicity is found usually in plants growing on acid soil while manganese-deficiency diseases are characteristic of many alkaline soils.

The manganese-toxicity disease of cotton is known as crinkle leaf (9, 10). Leaves become puckered, mottled, and variously distorted. Necrotic lesions appear between the veins and as the plants approach maturity, the leaves are slightly thickened, brittle, and ragged at the edges. Fasciation of branches follows. Chlorophyll deficiency and distortion extend to bracts and bolls, and yield and quality are decidedly reduced. Internal bark necrosis, common on Red Delicious apple trees, is due in large measure to manganese and possibly iron excesses (1). Continued use of ammonium sulfate on pineapple soils of Puerto Rico has led to a pH low enough to bring on manganese toxicity (6). It is suspected that chlorosis and necrotic spotting of soybean leaves on highly acid soils, where this crop is sometimes used as a cover crop because it is one of the more acid-tolerant legumes, are due to manganese toxicity since the symptoms correspond to those produced in controlled nutrient culture. Interveinal chlorosis and necrotic spotting of scarlet runner bean and in-curled margins of cauliflower have been shown in England to be manganese-toxicity diseases.

Aluminum and iron salts sometimes occur also in acid soils to the extent that they are toxic.

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Potash-deficiency Diseases

Not a great deal is known about the function of potassium in the metabolism of plants. It does not enter into the compounds ordinarily recognized in the protoplast of the cell or in the cell membrane, but relatively large amounts are needed, and the potassium salts in some way are retained tenaciously by the living plant tissue. Much has been learned of the function of potassium by what follows when this element is lacking or deficient.

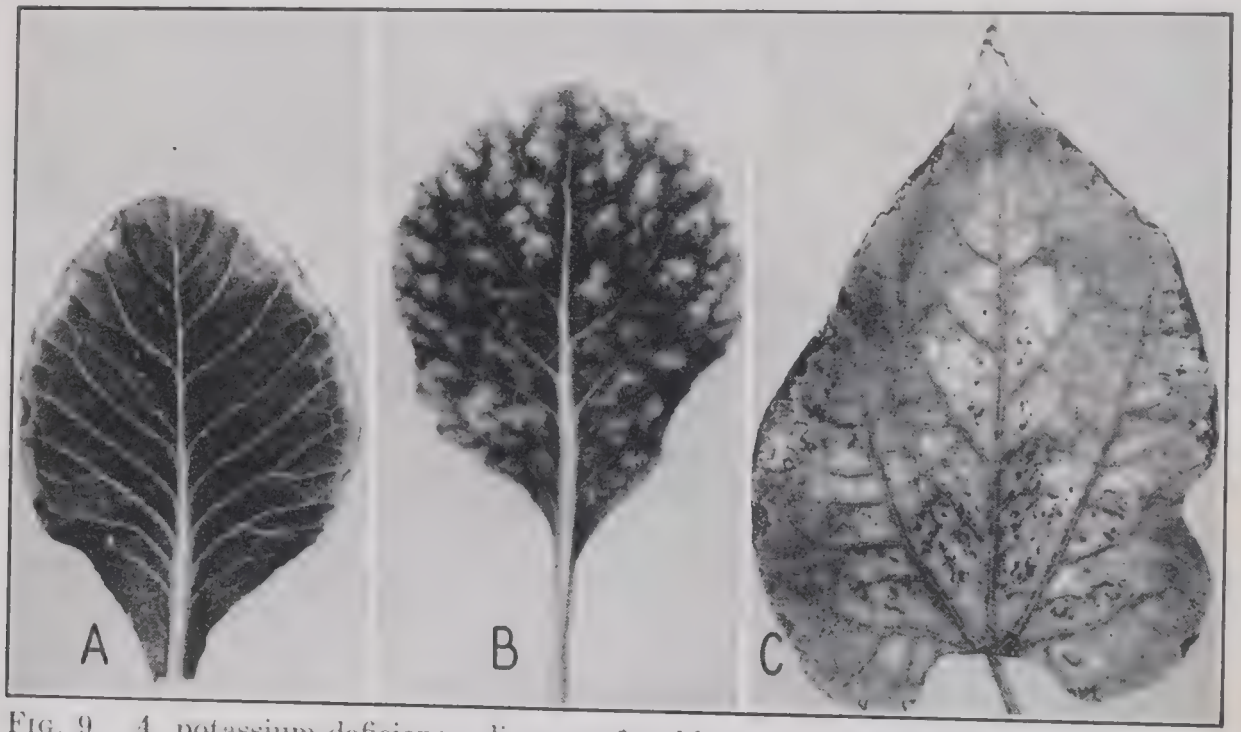

Fig. 9. *A*, potassium-deficiency disease of cabbage; *B*, iron-deficiency disease of cabbage; *C*, magnesium-deficiency disease of bean.

In many plants, such as tobacco, cabbage, cucumber, tomato, grape, apple, and prune, the first symptoms of potassium deficiency are mottle and chlorosis of the older leaves, particularly near the margins (Fig. 9). This condition may advance to include the interveinal tissue of the entire leaf, while the marginal tissue turns brown, dies, and becomes brittle. Necrotic spots in the inner portion of the leaf blade may follow. In corn plants the leaves become yellow, or streaked with yellow, while the edges fire and the tips of the ears do not fill. In potato the first sign of potash hunger is the appearance of abnormally dark green foliage; this is followed by yellowing or bronzing, beginning at the leaflet margins. In alfalfa and clovers early signs of potash deficiency are small white spots scattered over the leaf blade or localized near the margins. In all these cases the secondary effect is reduced productivity and lower quality of the product.

Potash deficiency is a very common disease in cotton, where it is often called "cotton rust." Here again the bronzing and marginal burning of the leaves are the first signs, and the leaves finally become reddish brown and die, while the bolls become dwarfed, fail to open, and contain fiber of poor quality. Potassium deficiency in citrus produces quite distinct symptoms. Instead of the marginal chlorosis and burning so characteristic of many species, there occur twisting and crinkling of the leaves; weak, flaccid, S-shaped lateral shoots; veinyellowing, yellow spots, markings, and stipplings on the leaves.

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Phosphorus-deficiency Diseases

Phosphorus is contained in some of the amino acids and proteins of the plant cell. Many soils are deficient in this element, and phosphorus starvation is of common occurrence. The signs of phosphorus deficiency are not easy to define since they consist chiefly in retarded growth and maturity. Sugars accumulate in the tissue, and in some plants this promotes the formation of purple pigments. Reddening or purpling of leaves is a common sign of phosphorus deficiency when the plant is capable of responding in this way. Some strains and species do not have the property of producing these pigments. In cotton, dwarfing and dark green foliage occur. Peaches are particularly sensitive to phosphorus deficiency, the vigor of trees often becoming decidedly reduced. The older leaves take on a dark green color, and this is followed by gradual purpling of the veins on the lower sides of the leaves and of the petioles, while the upper sides of the leaves become bronzed and tanned in color. The newest leaves become more erect than usual, while somewhat older leaves tend to curl downward at the margins and at the tips.

On calcareous soils in the Red River valley of North Dakota chlorotic dieback of flax develops in cool wet weather. This disease consists in chlorosis of leaves and killing of the terminal bud. In dry cool weather the

cotyledons have a greenish-bronze cast, and the leaves are stunted and flecked. Since application of phosphates tends to correct the disease, it is probably due, in part at least, to phosphate deficiency.

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Nitrogen-deficiency Diseases

Nitrogen is essential in all protein formation, and since it is such a vital element in many plant processes, its lack is the common cause of deficiency diseases. In practically all cases the first sign of nitrogen shortage is a change from the normal green color to a lighter or more yellowish green. Protracted starvation leads to intensification of yellow shades and finally to drying up of the leaves. This response is common among most species of plants. In consequence, growth is retarded in proportion to the degree of nitrogen deficiency. Nitrogen deficiency can usually be corrected by prompt application of readily available nitrogenous fertilizers.

Calcium-deficiency Diseases

Calcium acts to neutralize some of the plant acids in the protoplast, such as oxalic acid, and in some way maintains a balance with magnesium, potassium, and boron which is essential to the normal physiological processes of the plant. It appears to be an essential part of the nucleus, and its absence profoundly affects cell division. The common opinion that it is essential to the formation of the middle lamella is not upheld by the work of Sorokin and Sommer (4). Calcium salts, particularly the hydrate and carbonate, are applied extensively to the soil to bring about the best reaction for plant growth, largely through the effect of such reaction on the availability of other nutrients and upon the soil flora. Calcium, however, is needed by the plant as a nutrient, and, contrary to a common opinion, many soils are deficient in this element; such deficiency is not necessarily correlated with high acidity since acid soils differ in the amount of calcium available to the plant. Calcium is not readily transferred within the plant from old tissue to newly forming leaves and shoots.

The development of calcium-deficiency-disease symptoms is influenced by the supply of the other elements mentioned above which interact with it in cell metabolism. Reference to one type of such interrelation is made under Boron-deficiency Diseases (p. 83). Because of this interrelation,

distinct calcium deficiency is not readily discerned in the field. Much of the available knowledge about its effects has been secured from controlled-nutrient cultures.

The first symptom of calcium deficiency on tobacco is a lighter than normal green color and a distinctive downward curl of the tips of the leaves around the terminal bud. Necrosis of the tissue begins at the margins of the leaves and proceeds until the whole leaf dies, or localized necrosis followed by normal growth leads to leaf malformation. In potato a light green band along the margins of young leaflets is followed by necrosis or by wrinkling due to lack of uniform growth. Necrotic spots may appear in the pith tissue of the tuber. In tomato, chlorosis of the younger leaves occurs, followed by terminal necrosis and by flabbiness of the plant as a whole. In peas red patches appear in the leaflets near the midrib, discoloration at the margin developing last. When pea plants are grown in complete or nearly complete absence of calcium, the stems die promptly and roots are killed at the tips, resulting in a very reduced root system (1). In apple and peach, calcium deficiency appears first as short stubby roots, before signs develop in the leaves. The aerial symptoms in apple may consist only in reduction in size of leaf and shoot. When acute symptoms occur, they consist in chlorosis, followed by necrosis at the margins and sometimes in the interveinal regions. In peach trees aerial symptoms appear promptly, first on the basal leaves of the twig as necrotic areas along the midrib or at the tip. With extreme deficiency, acute dieback of twigs develops.

Withertop of flax in Australia consists in a sudden bending and loss of turgidity of the stem 2 to 4 in. below the tip, followed by necrosis at the point of bending and death of the tip. This is followed by axillary side-branch development. It occurs on plants 12 to 15 in. high or higher. Waterlogging of the soil favors the disease, and application of lime or gypsum corrects it (2).

Wilting and chlorosis of the leaves accompanied by stunted root growth of *Philodendron* cuttings were found to be due apparently to calcium deficiency (5).

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Magnesium-deficiency Diseases

The content of magnesium in plant tissue is ordinarily less than that of calcium. It is contained in the chlorophyll molecule and is regarded as an essential part of phosphate metabolism. Magnesium is mobile in the plant, and when the supply in the soil is low, it is drawn from older leaves to young actively growing ones. Until relatively recently it had been assumed that enough of this element is available in most soils to provide adequately for the needs of plants, but this is now known to be false. The soils of large geographic areas, such as the coastal plain of the Atlantic seaboard, are deficient in magnesium, having been derived from rocks which contain calcium to the nearly complete exclusion of magnesium. In other areas where soils are derived from dolomitic limestone, magnesium is abundant.

Sand drown is a magnesium-deficiency disease of tobacco long known in Atlantic seaboard states on deep sandy soils. Symptoms appear on plants in midseason as a progressive loss of normal green color of the lowest leaves. Unlike that of potassium deficiency, the chlorosis associated with magnesium deficiency occurs throughout the parenchyma tissue of the leaf blade rather than predominantly at the tip or margin. The leaves are progressively affected from the lower part of the plant upward. In corn it appears on old leaves first, as yellowish to white streaks extending the entire length of the leaves. Potatoes are affected by magnesium deficiency throughout the Atlantic coastal plain. The lower leaves become lighter in color, beginning at the tips and margins and progressing toward the midrib. Chlorotic tissue of the leaf blade of many plants becomes occupied by small brown necrotic spots (Fig. 9). As the magnesium is withdrawn from older leaves to younger ones, the disease progresses upward, and leaves tend to roll upward while the entire plant becomes stunted. Many vegetables show the same general type of chlorosis and stunting described above, practically always starting with lower leaves.

In New Jersey, magnesium deficiency causes older leaves of peach to turn dark green and pass into marginal, then general, chlorosis and necrotic spotting. Defoliation follows, fruit-bud formation is curtailed, and young trees fail to overwinter. Magnesium deficiency is seldom found on apples in the United States, but it is a serious disease in New Zealand. On citrus this disease occurs chiefly in Florida, where it is a major problem. Yellow blotches along the midrib of older leaves expand to occupy the entire leaf. Movement of magnesium from leaf to fruit is followed by leaf drop. Defoliation is particularly severe in orange.

Soils deficient in magnesium are corrected by liberal applications of dolomitic limestone. When heavy dosages of lime are undesirable, as on potato soils, magnesium sulfate may be used, or the use of dolomitic

hydrate of lime in Bordeaux mixture spray may suffice. As crops show symptoms, magnesium sulfate may be sprayed upon them as a means of rapid control.

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Sulfur-deficiency Diseases

Sulfur is contained in amino acids which become a part of certain proteins. In some families of plants, such as onions and crucifers, sulfur-containing essential oils are characteristic. Sulfur-deficient soils are not common, and although little emphasis is placed upon it as a component of fertilizer, it is often included as ammonium sulfate, superphosphates, or gypsum. Sulfur deficiency seldom occurs in the field. Rarely it is found to cause yellowing and dwarfing of cotton where sulfur-containing fertilizers have not been applied for a long time.

Iron-deficiency Diseases

A relatively small amount of iron is needed by plants, but its absence causes very acute disease symptoms. As with other trace elements, its function is not well understood, but the type of symptoms which follow removal of iron from the nutrient shows that it is concerned with the process of photosynthesis. Very acute yellowing, usually with little or no mottle pattern, is the almost universal result of iron deficiency (Fig. 9).

Chlorosis associated with calcareous soils is sometimes the result of the conversion of available ferrous salts to unavailable ferric salts in the alkaline soil solution. In acid soils iron is usually available, but it may be made unavailable by an excess of soluble phosphates. When acute iron chlorosis occurs on trees or vines, it can usually be corrected by the application of ferrous sulfate to the soil or to the foliage as a spray.

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Boron-deficiency Diseases

As early as 1900 stimulation of plant growth by addition of traces of boron to plant nutrients was observed. The first proof that this element is necessary for growth was furnished for corn in 1919 by Mazé in France. Warington in England in 1923 made a similar demonstration for certain legumes (29). In 1930 Mes in Sumatra showed that when tobacco was grown in a nutrient solution deficient in boron, there developed a disease similar to the top blight commonly observed but hitherto unexplained. Brandenburg (2), in Holland, in the next year noted that when sugar beets were grown on liquid nutrient practically free from boron, a disease developed which coincided in symptoms with that known as heart and dry rot. This disease had been known in Europe for many years. It consisted in distortion and necrosis of the heart leaves (heart rot) and an internal breakdown of the fleshy root tissue (dry rot). Further study confirmed that this was a boron-deficiency disease, and it was corrected by applying borax (sodium tetraborate) to the soil. Since 1930 puzzling diseases of many plants have been found to be boron-deficiency diseases. Furthermore, with the plant symptoms as indicators more information has been gained as to the function of boron in the plant. In many earlier plant-nutrient studies, boron deficiency was not recognized although the element was not consciously added to the nutrient. Probably the traces needed were applied as an impurity in the nutrient salts or were dissolved from the containers.

Many soils are now known to be deficient in boron (31). This condition may have arisen from leaching, or it may have been brought about by fixation of boron in a form unavailable to plants. There are different views as to the methods whereby boron becomes fixed in soils (14, 15). It is commonly observed that high-lime soils are deficient in available boron, and that boron becomes less available and presumably temporarily fixed during protracted periods of drought. Boron is also fixed rapidly in plant tissue and is not translocated from one part of the plant to another after it has become fixed (2).

The interaction of calcium, potassium, and boron in the nutrient substrate has an important bearing on the development of boron-deficiency or boron-toxicity symptoms. Reeve and Shive (20) studied this problem on tomato in sand nutrient cultures. At any given level of boron in the substrate the boron content of the plant increased with increase in concentration of potassium in the substrate. At low boron levels increase of potassium led to deficiency symptoms; at high boron levels it led to decrease in toxicity. In this latter relation calcium had an effect opposite to that of potassium. There is a close relation between calcium and boron in response of the plant to high or low levels of the latter. Potassium seems to affect the response of the plant to boron through its effect upon the processes involved in the absorption and accumulation of calcium. Lorenz (11) in a study of table beets also found the interaction of boron and calcium opposite to that of boron and potassium. Boron increments were most efficient in increasing growth at high levels of potassium and at low levels of calcium in the substrate. He suggested that while boron may influence the absorption of calcium and potassium, it functions chiefly in its effect upon their utilization. Jones and Scarseth (10) in a study of Indiana soils concluded that each plant species has a specific need for calcium and boron and that normal growth occurs when a certain balance exists. On acid soils plants with low calcium intake usually have a low tolerance for boron, and on very acid soils borax in small amounts is commonly toxic. On alkaline soils plants require more calcium and more boron; hence boron deficiency is more common. Likewise more borax can be added without injury.

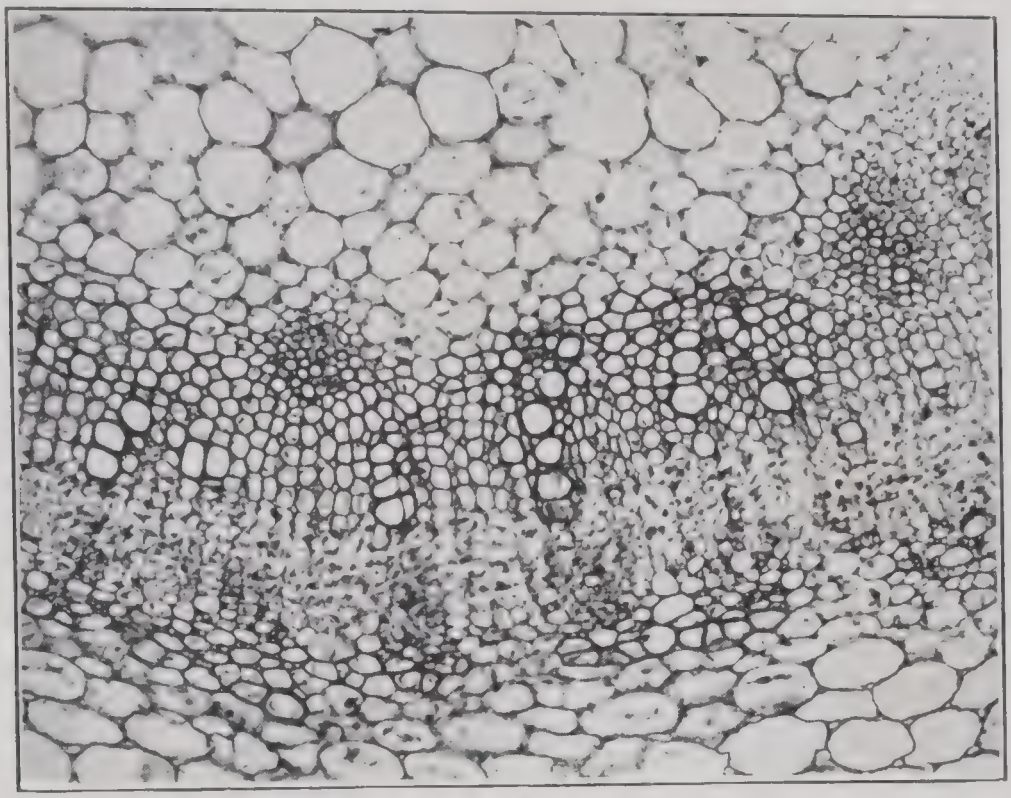
Scripture and McHargue (22) found that in alfalfa grown in sand culture with boron-deficient nutrient, sugars and soluble nitrogen accumulated. This agrees with the result of Wadleigh and Shive (25), who noted in a microchemical examination of boron-deficient cotton that the acidity of the cell sap increased, the ammonium nitrogen increased, the oxidation rate of sugars decreased, and the aminization of carbohydrate derivatives was reduced. Thus the production of proteinaceous compounds essential for protein metabolism was inhibited. Lorenz (11) suggested from a study of table beets that boron deficiency prevented the formation of propectins in the cell walls.

It is generally found that the chief anatomical response to boron deficiency is in the meristematic regions of the plant. Warington (30) recorded the chief features in boron-deficient broad-bean plants as hypertrophy of cambium cells followed by degeneration and disintegration in this and other tissues and lack of normal differentiation. Sommer and Sorokin (24) found a disturbance in the development of cells, leading to hyperplasia, hypertrophy, and abnormal differentiation in roots of

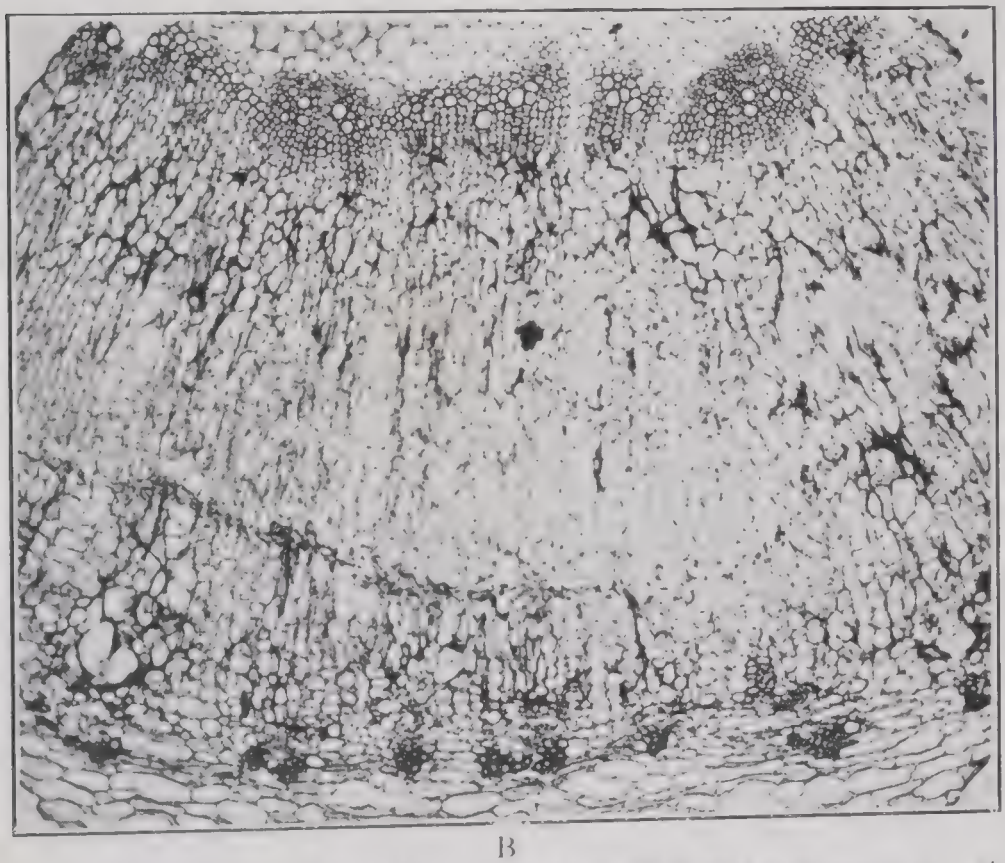
boron-deficient pea plants. Jolivette and Walker (9) compared boron-deficient and normal roots of beet and of cabbage. The greatest degree of response was in the active regions. In growing beet roots this is in the secondary cambium rings laid down in the pericycle. These do not have the same level of activity at the same time, and consequently boron-deficiency symptoms appear in some more pronouncedly than in others. The first signs of the disease are in phloem cells, which are filled with dense materials. In xylem cells intercellular brown deposits appear, and cells begin to show signs of abnormal size and shape. These are the beginnings of necrotic areas which later become visible in size, at which time they consist of patches of dead cells, often walled off from the rest of the tissue by a zone of rapidly dividing cells. The cambium cells in this region either continue to divide with little differentiation into phloem and xylem, or they develop into giant parenchyma cells. A similar distortion of vascular elements occurs in leaf petioles and in the stems of seed plants. In roots of cabbage seedlings there is excessive cell division with inhibition of normal differentiation into xylem and phloem (Fig. 10). Reed (19) has described the physiological and biochemical processes which are characteristic of cells in boron-deficient tissues.

Boron appears to become fixed rapidly in the plant tissue and is not transported readily after fixation occurs. Boron can be applied to soil as borax (sodium tetraborate) and as boric acid. It is usually applied to soil or sprayed upon plants as borax. Approximately 8.8 lb. of borax or 5.8 lb. of boric acid are required to provide 1 lb. of boron. The requirements depend upon the soil and the crop concerned. An excellent summary of the various symptoms of boron deficiency on important crop plants is found in "Boron in Agriculture," published by the Pacific Coast Borax Company. The diseases of a few representative crops will be described briefly.

Boron-deficiency Disease in Beets. The heart and dry rot of sugar beet is very common in Europe (2), in the United States (28), and in other parts of the world where this crop is grown. One of the most important symptoms is a necrotic crosshatching of the tissue on the inner concave surface of the leaf petiole. This is a fairly certain sign of boron deficiency when other evidence is lacking. The young unfolding leaves fail to develop normally and eventually become necrotic and die. If boron deficiency continues, dormant buds in the axils of older leaves may grow and in turn die. As this process continues, the heart of the beet top becomes a rosette of more or less desiccated leaves. Within the fleshy root, breakdown occurs in one or more of the cambium rings. When these necroses occur near the surface, the breakdown may lead to a canker, and secondary organisms may enter. Where this disease is likely to occur, it is controlled



A



B

FIG. 10. Pathological histology of boron deficiency in cabbage seedling. *A*, cross section of stem of seedling grown in a complete nutrient; *B*, cross section of stem of seedling of same age grown in the same nutrient without boron. Note excessive cell division and reduced amount of differentiation of cambium cells into xylem and phloem. (*After Jolivette and Walker.*)

readily by broadcasting 10 to 20 lb. of borax per acre and disking in thoroughly before sowing the crop.

In table beets and chard similar symptoms on the foliage occur (26, 28). In the former the internal breakdown of the root known as internal black spot is often more pronounced and of greater importance than in sugar beet since it causes roots to be of questionable value as food (Fig. 11). Much loss in the raw product and an increase in cost of operation are occasioned where table beets affected with boron deficiency are used for

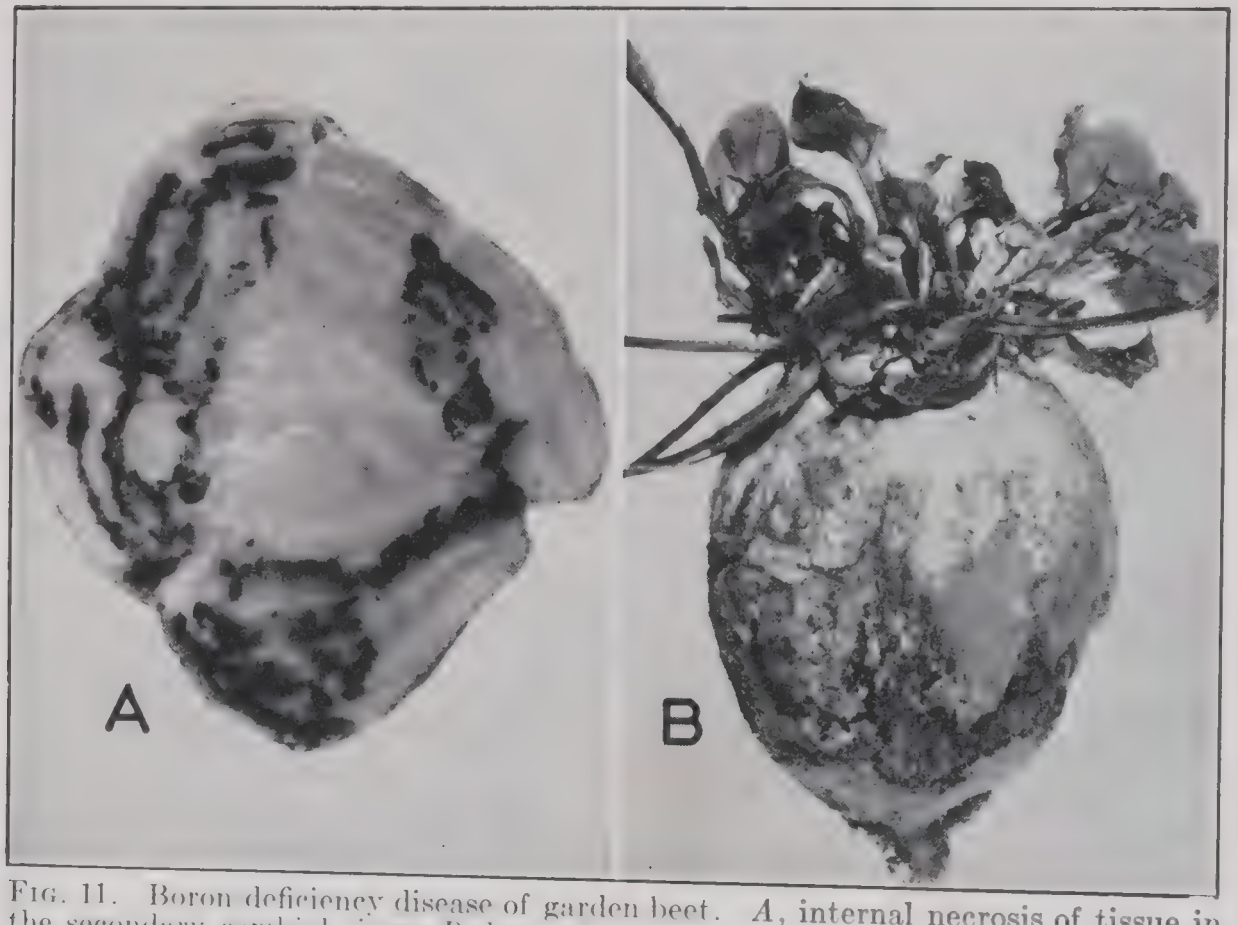


FIG. 11. Boron deficiency disease of garden beet. *A*, internal necrosis of tissue in the secondary cambial rings; *B*, leaves become stunted, and dormant buds of the crown are stimulated to form small distorted leaves. Internal necrosis near the exterior of the root leads to collapse of the outer tissue to form cankers.

commercial canning. In many areas where beets are grown on neutral to slightly alkaline soil relatively high in organic matter, it is necessary to apply 40 to 60 lb. of borax per acre to reduce internal black spot to a negligible quantity. The top symptoms, including necrosis, may be the indirect effect of the disturbance of normal function and development of the vascular tissue.

It is evident that boron is essential to normal growth and differentiation. Numerous experiments have shown that when borax is applied to beets, increased yields follow even though deficiency symptoms were not observed in untreated controls. This may be due to the fact that the

microscopic effects of boron deficiency described above result in reduced growth and lower yields without advancing to the point where macroscopic symptoms develop. Under such circumstances boron applications affect growth sufficiently to increase yield.

Boron-deficiency Disease of Apple (3, 32). In British Columbia, in northeastern New York, and in many other apple-growing regions, a series of disease manifestations known as internal cork and drought spot of the fruit and rosette and dieback of the tree reached serious proportions by 1920. In 1934, soon after the discovery of the relation of boron to top blight of tobacco and heart rot of sugar beet, McLarty in British Columbia demonstrated that the diseases were corrected by feeding boron to the trees.

Internal cork consists in light brown spots of dead tissue within the flesh of the apple. When necrosis is severe early in the growth of the fruit, the latter may be markedly deformed or noticeably roughened on the surface. Drought spot occurs when lesions are on the surface, causing russeting and cracking. When boron deficiency is acute, the leaves and new wood are affected. The leaves turn yellow and brown, curl, and eventually die and drop. Sometimes stiff, thick, brittle leaves remain on the tips of the twigs, causing a rosette. Twigs may die from the tip back to the old wood, and as new growth appears, it dies back. As this process continues, a witches'-broom effect appears.

Boron deficiency may be corrected by spraying trees with boric acid, but application of borax to the soil soon after growth starts in the spring is a more reliable treatment. About 6 oz. per tree up to 15 years of age, about 12 oz. up to 25 years, and 1 lb. over 25 years is the average recommendation.

Boron-deficiency Disease of Crucifers (9, 12, 23, 27). In 1933 it was shown by MacLeod in New Brunswick, Canada, that the common disease of turnip and rutabaga known as brown heart, or dark center, is a boron-deficiency disease. This is now known to be a common malady where these root crops are grown on boron-deficient soils. Water-soaked areas appear in the flesh of the root, becoming brownish in color. Roughened skin and yellowish leaves may occur, but these are not common symptoms. Dark discoloration and breakdown of the internal root tissue results in serious losses. Application of borax at the rate of 15 to 30 lb. per acre before sowing corrects the deficiency. In very high lime soil this type of application is sometimes not completely effective, presumably because the boron is fixed in the soil before the crop matures. In such instances two to four applications during the growing season of 2 per cent borax solution as a spray are more effective.

Cauliflower is sensitive to boron deficiency, and severe losses may

result. It was first noted in a severe form in the Catskill area of New York in 1934. The leaves around the curd become deformed. The curd takes on a brownish color, and this discoloration extends through the midribs of the curd to the main stalk, where, in addition to discoloration, splitting of the tissue results in a hollow discolored pith. Cabbage is less susceptible to boron deficiency than cauliflower, but when it is acute, discoloration and breakdown in the core of the head occurs (Fig. 12). This internal breakdown may be found as well in broccoli and kale. Ten to twenty pounds of borax per acre is usually adequate for the cole group of crucifers on heavy soils or those high in organic matter.

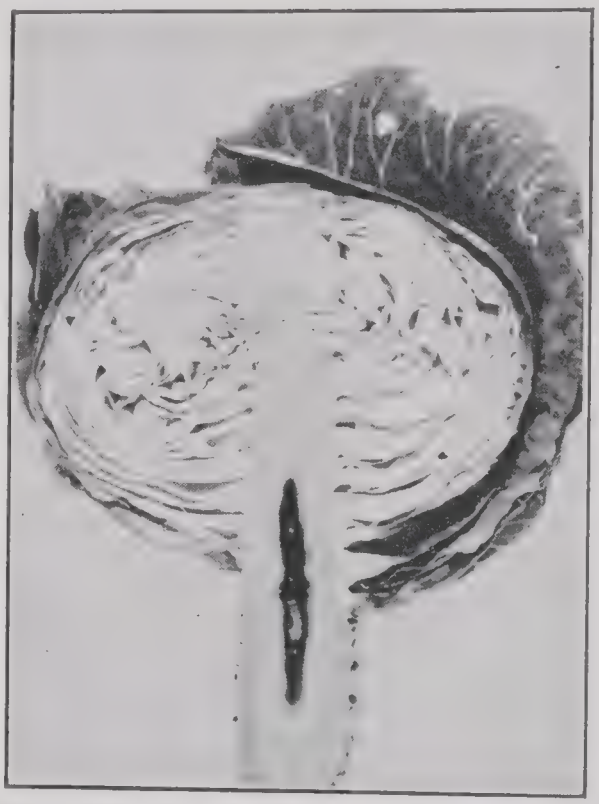


FIG. 12. Boron-deficiency disease of cabbage. Internal necrosis of the pith occurs as the plants approach maturity.

Boron Deficiency in Alfalfa. It is found that boron deficiency is a limiting factor in alfalfa production in some areas, *e.g.*, in Tennessee (17), Washington (1), Oregon (5), and Vermont (14). In some other regions the disease on alfalfa is not as severe, and response to borax is not as pronounced. Shortening of the internodes, accompanied by yellowing, reddening, and rosetting of the terminal leaves and sometimes killing of the growing point are the chief distinctive symptoms on alfalfa. These symptoms are difficult to distinguish from yellowing associated with the potato leaf hopper injury (4), except that the latter is not confined to the terminal parts. Ample boron sometimes improves the seed set of alfalfa (7, 17).

Other boron-deficiency diseases of importance are cracked stem of celery (18); internal brown spot and cracking of sweet potato (16); fruit pitting, chlorosis, and dieback of olive (21); chlorosis and root necrosis of radish (23).

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Manganese-deficiency Diseases

Manganese was known to be widely distributed in plants as early as 1889. Conclusive proof that manganese is an essential element for plant growth was published in 1922 by McHargue (10). Although the practice of adding manganese salts to the soil to correct the disease of oats known as gray speck was practiced widely before this, gray speck was not clearly shown to be a deficiency disease until 1929 by the studies of Samuel and Piper (14) in Australia. Since that time many crops have been shown to suffer from manganese-deficiency diseases.

As already pointed out, manganese salts tend to be converted to insoluble forms unavailable to the plant when the soil solution is neutral or alkaline. Where such soils prevail, manganese-deficiency symptoms appear on many crops. Alkaline muck soils are often low in available manganese. Alkaline soils are also commonly deficient in zinc, iron, and copper, and since deficiencies of these three elements cause symptoms which overlap with those of each other and those of manganese deficiency, a complex disease commonly occurs. Chlorosis with necrotic spots is the most common symptom of manganese deficiency, and these signs are accompanied by greatly reduced growth and consequent yields.

Gerretsen (6) studied the relation of soil bacteria to manganese and came to the conclusion that they were responsible in large measure for the conversion of available manganese to a nonavailable form. Eltinge (5) made a histological and cytological study of manganese deficiency on the cells of the tomato. Chloroplasts are affected first. They become yellow-green in color, lose starch, become vacuolated, granular, and finally they disintegrate. Palisade cells are smaller; leaves and stems are thinner. The xylem is less fully developed, and a certain amount of plugging takes place.

The gray-speck disease of oats, which consists in mild chlorosis of the leaves with characteristic gray necrotic spots, has long been known as a serious disease on many soils in Europe and in Australia. It is also recognized rather widely in the United States. Marsh spot of garden pea

is another disease long known in Europe, occurring particularly on reclaimed marsh soils (9, 13). The chief symptom is a necrotic spot in the cotyledon of the seed at the succulent edible stage. This has been shown to be a manganese-deficiency disease. Manganese chlorosis is a serious disease of beans on uncorrected muck soils of Florida. Chlorosis from the same cause occurs on citrus in California and Florida. Pahala blight of sugar cane in Hawaii is a manganese-deficiency disease. English walnuts in California show interveinal mottle, bronzing, and scorching of leaves. Tomatoes, beets, and spinach may suffer from manganese deficiency also.

This group of diseases is readily controlled by application of relatively small amounts of manganese sulfate or chloride to the soil (up to 100 to 200 lb. per acre) with the fertilizer. These salts may also be applied as a spray to the growing crop.

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Zinc-deficiency Diseases

The first attention to zinc deficiency as a cause of plant disease came about 1930. In California it was found that apple rosette, little leaf of stone fruits, and mottle-leaf of citrus were corrected by the application of zinc salts (3, 4). In apple, leaves of current-season twigs remain small and stiff and form terminal rosettes. Branches arise from below the twigs. There is some mild chlorosis. Little leaf and rosette, much like that on apple, also form on peaches. Mottle-leaf is common on citrus in California and in Florida. On mature leaves varied degrees of chlorosis occur. New leaves are narrow, and twigs are dwarfed. The tree takes on an abnormal upright bushy appearance. The fruits are small and thick-skinned. Pecan rosette, a disease known for a long time in the southern and southwestern states, is also a zinc-deficiency disease (1, 5).

On alkali muck soil, particularly recently burned muck, zinc deficiency of truck crops occurs. Squash, mustard, tomato, potato, and beans are sensitive. The symptoms on the leaves are a chlorotic mottle with necrotic spots and dwarfed development.

In central and northwest Florida, on acid mineral soils, a disease of corn known as white bud is due to zinc deficiency. This is a chlorosis which appears soon after the seedlings emerge. Light yellow streaks appear between the veins. This yellow striping continues to appear on newly unfolding leaves, and some white patches devoid of chlorophyll may occur. Growth is stunted markedly (2).

Histological study of mottle-leaf of citrus shows elongation of the palisade cells. Plastids are few in number and small; they tend to aggregate. The cytoplasm is in a thinner than normal layer, and the nucleus is abnormal. The xylem parenchyma tends to be woody, and calcium oxalate crystals are greater than normal in number (7).

Zinc deficiency is corrected on trees by spraying with zinc sulfate. Soil applications are sometimes more satisfactory for annual crops.

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Copper-deficiency Diseases

In 1914 Brenchley noted that very small increments of copper stimulated plant growth, but in larger amounts it was toxic. In 1931 Sommers reported results of experiments which proved copper to be essential. Lipman and MacKinney (5) reported similar results simultaneously. Dieback of citrus had been controlled with copper sulfate by Grossenbacher in Florida in 1916. Ritzema Bos by the use of copper sulfate also controlled a disease on oats characteristic of reclaimed soils, and therefore called "reclamation disease." In 1927 Allison *et al.* (1) in Florida and Felix (4) in New York controlled a severe chlorosis of lettuce on muck soils by the application of copper sulfate in small amounts as a soil amendment or as a spray. There are now numerous diseases in crops which are known to be due to copper deficiency.

Withertip, or summer dieback, of apples in Australia is due to copper deficiency (3). Brown spots appear on terminal leaves, and they gradually wither and fall. The branch then dies back 3 to 12 in. Trees become stunted and bushy from inability to develop leader branches. Cupping and chlorosis of terminal leaves are the chief symptoms in tung. Dieback, or exanthema, of pear consists in brown discoloration and necrosis of tips of shoots and of terminal leaves (6). Annual dying back of shoots results in a bushy, stunted tree.

In reclamation disease of oats white papery leaf tips develop; the glumes are white and loose. Yellow tip is found also in barley, red clover, broad bean, alfalfa, pea, and rutabaga.

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Molybdenum-deficiency Diseases

Molybdenum was shown to be essential for normal growth of tomato seedlings by Arnon and Stout (4) in 1939. Whiptail of cauliflower, which consists in leaf malformation and unsatisfactory curd production, has been observed in Long Island for many years, as well as in some other parts of the world. In Long Island the disease is prevented by application of lime to the relatively highly acid sandy soils where cauliflower is grown. The same disease in New Zealand has responded to application of 5 to 20 lb. of ammonium molybdate per acre (6). The application of lime appears to increase the availability of molybdenum in the soil. In some locations in Australia legumes and cauliflower respond favorably to the application of molybdenum salts (1 3, 9). Molybdenum appears to be essential for symbiotic nitrogen fixation and for optimum activity of *Azotobacter*. Wilson and Waring (9) suggest that molybdenum acts as a catalyst in the reduction processes in the cauliflower plant, probably in the reduction of nitrates.

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CHAPTER 4

BACTERIAL DISEASES

Since the first recognition of a bacterium as a pathogen of plants by Burrill in 1878, many bacterial diseases of plants have been described. In the sixth edition of "Bergey's Manual of Determinative Bacteriology" (1948), 159 species are listed as plant pathogens. In Chap. 2 we have related some of the landmarks in the formative period of bacterial plant pathology.

The bacterial plant pathogens, except for species of *Streptomyces*, are nonsporeforming rods. Their differentiation on the basis of morphology is difficult, and in early schemes of classification it was based chiefly on the number and location of flagella. The position of flagella is indicated by the following terms: monotrichous, one flagellum at one end; lophotrichous, more than one flagellum at one end; amphitrichous, one or more flagella at each end; peritrichous, flagella scattered over the outer surface. Even this determination was uncertain because of variability in this character in pure culture and the difficult technique involved in staining flagella. Since morphology proved to be inadequate, differentiation on the basis of physiological characters had to be employed. The most common characters used include kinds of growth on agar plates and slants; gram stain; gelatin liquefaction; nitrate reduction; production of indole, hydrogen sulfide, and ammonia; growth in milk; acid and gas formation with various carbon sources. Here again many problems arise because of variability in cultures, difficulty in separating mixed cultures, distinction between variability and mixtures, differences between investigators in refinements of technique and interpretation of results.

The understanding of variability has improved greatly in recent years. The old idea of fixed and immutable species has been abandoned. The importance of nutrition and environment in perpetuating variants is now recognized more generally. If a variant is less suited to the environment and substrate than the original, it may soon disappear. On the other hand a variant, and often a nonpathogenic one, may grow more rapidly than the original on a given substrate and after a few transfers completely replace the latter. In nature, vigorous pathogens in association with the host may replace less vigorous forms, and selection in the direction of pathogenicity may be going on to provide considerable stability in this character, while on a given artificial substrate pathogenicity may decline. The fact that other taxonomic characters may change also makes characterization and classification the more difficult. Detailed descriptions of the

pathogens concerned in the diseases treated in this book are not given, largely because the reports of different investigators vary considerably for the reasons just mentioned. Access to such descriptions may be had by referring to "Bergey's Manual" (sixth edition) (2) and to Elliott's "Manual" (11).

Bacterial classification has gone through many changes since late in the last century, when literature on bacterial plant diseases began to appear. Therefore, the same species will be found under different Latin binomials. A comprehensive classification of bacteria was published by Migula (15), beginning in 1897. Another classification was proposed by Lehmann and Neumann (14) in 1896. Erwin F. Smith modified those systems somewhat for bacterial plant pathogens, and his system is followed in Elliott's "Manual" (11). Beginning about 1920 and continuing up to the present, a committee of the Society of American Bacteriologists has reworked bacterial classification and published "Bergey's Manual of Determinative Bacteriology." This manual has passed through six editions (1, 2), and it is to be expected that further revisions will be made in the future. It gives a list of synonyms for each species. Different investigators will undoubtedly prefer to follow different systems of classification. Some confusion, therefore, may be expected, but the amount will be less if one who publishes on a given disease, after deciding on the binomial name he chooses to use, gives also the one accepted in the system of classification most widely used in his country. In the United States this is the Bergey classification.

A brief key indicating the relation of genera containing plant pathogens to the classification scheme of "Bergey's Manual" (sixth edition), and the position of species treated in this book follows:

Class Schizomycetes Nägeli.

Order Eubacteriales Buchanan. Single rigid cells in chains or masses.

Family Pseudomonadaceae Winslow *et al.* No endospores; elongate straight or spirally curved cells; usually motile by polar flagella; usually gram-negative.

Pseudomonas Migula. Monotrichous or lophotrichous or nonmotile. Mostly gram-negative; 85 species listed in Bergey (sixth edition) as plant pathogens.

P. solanacearum E. F. Sm., incitant of southern bacterial wilt.

P. phascolicola (Burk.) Dows., incitant of halo blight of bean.

P. syringae v. Hall, incitant of bacterial brown spot of bean.

Xanthomonas Dows. Cells usually monotrichous with yellow, water-insoluble pigment; 47 species listed as plant pathogens in "Bergey's Manual."

X. campestris (Pam.) Dows., incitant of black rot of crucifers.

X. phascoli (E. F. Sm.) Dows., incitant of common bacterial blight of bean.

X. malvacearum (E. F. Sm.) Dows., incitant of angular leaf spot of cotton.

Family Rhizobiaceae Conn. No endospores; flagellate or nonmotile; usually gram-negative.

Rhizobium Frank. Cells usually capable of fixing free nitrogen when present in root nodules of legumes; 6 species listed in "Bergey's Manual."

Agrobacterium Conn. Small, short rods, typically motile; usually gram-negative; little or no gas or acid; gelatin not liquefied or sparsely so; 3 species (and 2 uncertain species) listed as plant pathogens in "Bergey's Manual."

A. tumefaciens (E. F. Sm. & Town.) Conn., incitant of crown gall of plants.

A. rhizogenes (Riker *et al.*) Conn., incitant of hairy root.

A. rubi (Hildebrand) Starr & Weiss, incitant of cane gall.

Family Corynebacteriaceae Lehmann & Neumann. Usually nonmotile rods, frequently beaded or banded with metachromatic granules; marked diversity in form; branching cells in a few species; usually gram-positive; aerobic or anaerobic; gelatin may be liquefied, nitrates may be reduced.

Corynebacterium Lehmann & Neumann. Slender, straight to slightly curved rods, with irregularly stained granules; sometimes club-shaped; snapping division forms palisade cell arrangement; usually nonmotile; gram-positive; 10 species listed as plant pathogens in "Bergey's Manual."

C. flaccumfaciens (Hedges) Dows., incitant of bacterial wilt of bean.

C. sepedonicum (Spieck. & Kotth.) Skapt. & Burk., incitant of ring rot of potato.

C. michiganense (E. F. Sm.) C. M. Jens., incitant of bacterial canker of tomato.

Family Enterobacteriaceae Rahn. Straight rods; peritrichous flagella, or nonmotile; gram-negative.

Erwinia Winslow *et al.* Usually require organic nitrogen for growth; species vary as to carbohydrate fermention, nitrate reduction, gelatin liquefaction; all plant pathogens; 22 species listed in "Bergey's Manual."

E. amylovora (Burr.) Winsl. *et al.*, incitant of fire blight.

E. tracheiphila (E. F. Sm.) Holland, incitant of bacterial wilt of cucurbits.

E. carotovora (L. R. Jones) Holland, incitant of soft rot of vegetables.

Order Actinomycetales. Organisms with elongated cells tending to branch, rudimentary hyphae, in some families producing conidia.

Family Streptomycetaceae. Conidia borne on sporophores.

Streptomyces Waksman & Henrici. Much-branched mycelium, conidia in chains; 2 species listed as plant pathogens in "Bergey's Manual."

S. scabies (Thaxt.) Waksman & Henrici, incitant of common scab of potato.

TYPES OF BACTERIAL DISEASES

The pathogenic bacteria relate themselves to their hosts in a variety of ways, and the effects on the plants differ widely. As yet none except species of *Streptomyces* are known to penetrate the cuticle directly, except when it has been broken or rifted by some means. The legume-nodule bacteria (*Rhizobium*) penetrate the noncuticularized root hair. Many species enter through stomata, some chiefly through hydathodes; others enter primarily through wounds. Intercellular invasion is common. Some species continue their action primarily in the intercellular spaces. Some gain access to the vascular system and become primarily vascular parasites. The bacterial diseases may be grouped as follows on the basis of the type of major effect on the host:

Soft Rots. Various bacterial decays of plant organs have been described in which the major effect is a slimy softening of the tissue. Most of these are considered here as one disease, **bacterial soft rot.** The bacteria usually become established in wounds and advance by the secretion of an enzyme which diffuses in advance and dissolves the middle lamellae of the cells. Plasmolysis and death of the cells follow, and the bacteria grow upon the dead plant tissue rather than upon the living cells.

Leaf Spots. Many bacterial diseases start with the invasion of stomata of leaves and other organs. Invasion of and multiplication within the substomatal chamber and the intercellular spaces lead eventually to necrosis of the invaded plant tissue. Various forms of leaf and stem blight follow, primarily in the parenchyma tissue. [See bean blights (p. 115); angular leaf spot of cotton (p. 112).]

Extensive Blights. A more extensive and rapid necrosis is to be found as in fire blight. The progress of the bacteria is often more rapid than in leaf spots, giving a scorched appearance to affected shoots.

Vascular Diseases. In some cases the leaf-spot organisms also invade the vascular system and become systemic. This is true with bean blights, which fall equally well into this and into the leaf-spot group. In other cases the invasion may be through stomata, water pores, or wounds, but the organisms promptly concentrate and multiply in the vascular system. This usually results in a stunting (*hypoplasia*) and often in an acute wilt. Common examples are black rot of crucifers, bean bacterial wilt, southern bacterial wilt, cucurbit bacterial wilt, and ring rot of potato. In all of those mentioned the bacteria become systemic in the larger xylem vessels. In later phases of the diseases the organisms may invade xylem parenchyma and other contiguous tissues, in which they are primarily intercellular. In one important disease, bacterial canker of tomato, the bacteria are

contained primarily in the phloem, rather than the xylem, during the early part of disease development.

Bacterial Galls. In the four groups just described the effect upon the host is primarily necrotic and or hypoplastic. Another group consists of those in which the primary effect of the pathogen on the host is the stimulation of cell division. This condition is known as **hyperplasia.** Some cells become abnormally large, a condition known as **hypertrophy.** The abnormal condition results in overgrowths, which usually take the form of galls on roots, stems, or other organs. Crown gall, which affects many species of plants, is the best known and most intensively studied disease in this group (p. 140). Others are olive knot, hairy root of apple, cane gall, beet pocket rot, oleander knot, and pea fasciation. The formation of root nodules in legumes, in response to nitrogen-fixing bacteria, is also a hyperplastic effect. While these organisms usually have symbiotic relation with the host, they may at times be pathogenic.

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BACTERIAL SOFT ROT OF VEGETABLES

Bacterial soft rot is one of the most destructive diseases of vegetables in storage and transit. In blackleg of potato, now regarded as a phase of the same disease, the growing plant is affected, as well as the tubers before and after harvest. The disease was studied intensively in the years just preceding and following 1900 by L. R. Jones (5, 6) in the United States, by Harrison in Canada, by Appel in Germany, and by van Hall in Holland.

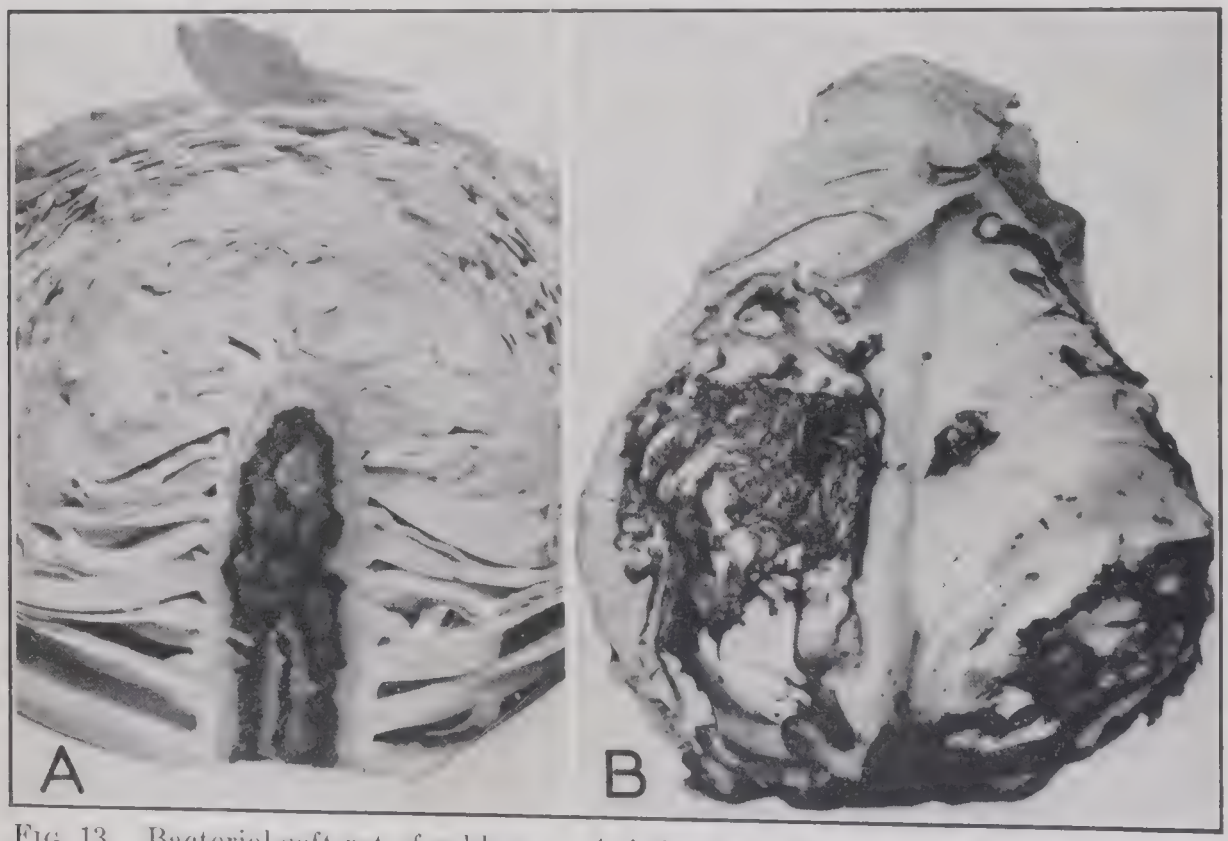

Fig. 13. Bacterial soft rot of cabbage. *A*, infection following invasion of the lower leaves by black-rot bacteria resulting in soft rot of the core of the head; *B*, infection resulting from penetration through surface wounds. (*B*, *courtesy of U.S. Department of Agriculture.*)

The association of the causal organism with the seed-corn maggot fly was worked out by Leach and first reported in 1926 (7).

Symptoms. The common name of the disease arises from the characteristic soft decay of the fleshy tissues. Carrot roots, potato tubers, onion bulbs, and fleshy petioles of celery are reserve storage organs, and hence the cells are in a semidormant or dormant condition, and the content of carbohydrate and nitrogenous materials is high. When soft rot affects them, the tissue softens; it becomes watery or slimy in consistency, and as the rot progresses, the water extrudes; or in a dry atmosphere water is lost rapidly by evaporation (Fig. 13). Some tissues, such as those of onion, cabbage, and potato, give off an offensive sulfurous odor, which may be due in part to the action of secondary bacteria.

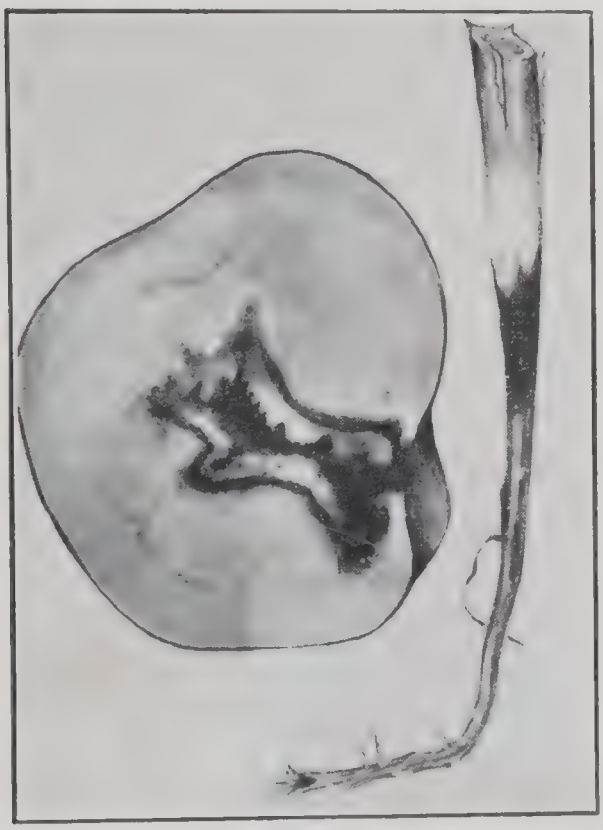

Fig. 14. Blackleg of potato.

When the seed tubers of potato are affected in the soil, the same type of decay follows. If the soil is moist for a protracted period, the shoots arising from the seed tuber are similarly affected, the cortical tissue becoming watery and the entire shoot wilting. If the soil moisture is reduced as the disease progresses, the base of the shoot develops a blackened, shriveled cortex and its growth is stunted, but it is not promptly killed (Fig. 14). The leaflets become reddish in color at the tips; the branches become stiffened and more upright than normal; and the entire shoot gradually becomes pale and yellowish. It is the latter phase of the disease on potato which is commonly known as blackleg.

The Causal Organism. *Erwinia carotovora* (L. R. Jones) Holland, 1923. The soft-rot group of bacteria contains organisms which are closely related in their morphological and physiological characteristics and in their serological affinities. Investigators differ as to whether they should all be regarded as members of the same species. Some regard them as variants of the same organism. The following resemble or are the same as the one described by L. R. Jones in 1901 as *Bacillus carotovorus*:

Bacillus hyacinthi septicus Heinz, 1889.
Pseudomonas destructans Potter, 1899.

Bacillus phytophthorus Appel, 1902.
Bacillus atrosepticus van Hall, 1902.
Bacillus omnivorus van Hall, 1902.
Bacillus aroideae Townsend, 1904.
Bacillus cepivorus Delacroix, 1905.
Bacillus solanisaprus Harrison, 1906.
Bacillus melonis Giddings, 1910.
Bacillus melanogenes Pethybridge & Murphy, 1910.
Bacillus cyprepedii Hori, 1911.
Bacillus dahliae Hori & Bokura, 1911.
Bacillus apiovorus Wormald, 1913.
Bacterium carotovorum (L. R. Jones) Lehmann & Neumann, 1927.
Pectobacterium carotovorum (L. R. Jones) Waldee, 1945.

Disease Cycle. Wounds are the most common avenues of infection. Harvest bruises, freezing injury, and insect wounds are frequent predisposing factors. The organism lives over in soil and in decayed refuse. Abundant moisture at the surface of the tissue, where wounds are present, is essential for invasion. Penetration through lenticels of freshly harvested potatoes has been observed (16). After infection has taken place, fairly high relative humidity is essential for progress of the disease. When decaying roots or tubers are placed in a dry atmosphere, the rotted tissue dehydrates rapidly, and further advance of the disease may be checked completely.

Several species of maggot fly are known to carry the organism. Adult flies lay eggs on decaying vegetable material, and as the eggs hatch, the larvae feed upon such debris, ingesting bacteria including the soft-rot organisms. There is good evidence that such organisms are essential to the normal development of the larvae. The intestinal tract thus becomes infested, and the pupa and the adult fly are each internally contaminated. Infested adults lay eggs in the soil just above planted potato seed pieces, and on maturing carrot, onion, cabbage, or turnips. As such eggs are laid, they are smeared with soft-rot bacteria. Mouth parts and intestines of the young larvae become contaminated. As they feed on potato seed pieces, or fleshy organs of vegetables, they carry bacteria and infect the wounded plant tissue. The gnawing action of the larvae provides a particularly favorable and efficient means of infection. The larvae penetrate any cork barriers the plant may form. Thus the insect becomes a very important vector of the causal organism. The relation of the insect to the organism has been worked out by Leach (7, 12), Johnson (4), and Bonde (1, 2).

The bacteria produce an enzyme which is capable of dissolving the middle lamella of the host tissue. This process was studied in detail by Jones

(6), whose work was a pioneer investigation in bacterial enzymes associated with pathogenicity. He referred to the enzyme as pectinase, but since it actually acts upon propectin in the middle lamella, it is, according to current terminology, referred to as **middle-lamella propectinase**. The action of this enzyme is to dissolve the middle lamella and thus to loosen the cells from one another (Fig. 15). The by-products of bacterial growth also set up exosmosis of liquid containing sugar and salts from within the cells to the intercellular spaces, where it undoubtedly serves

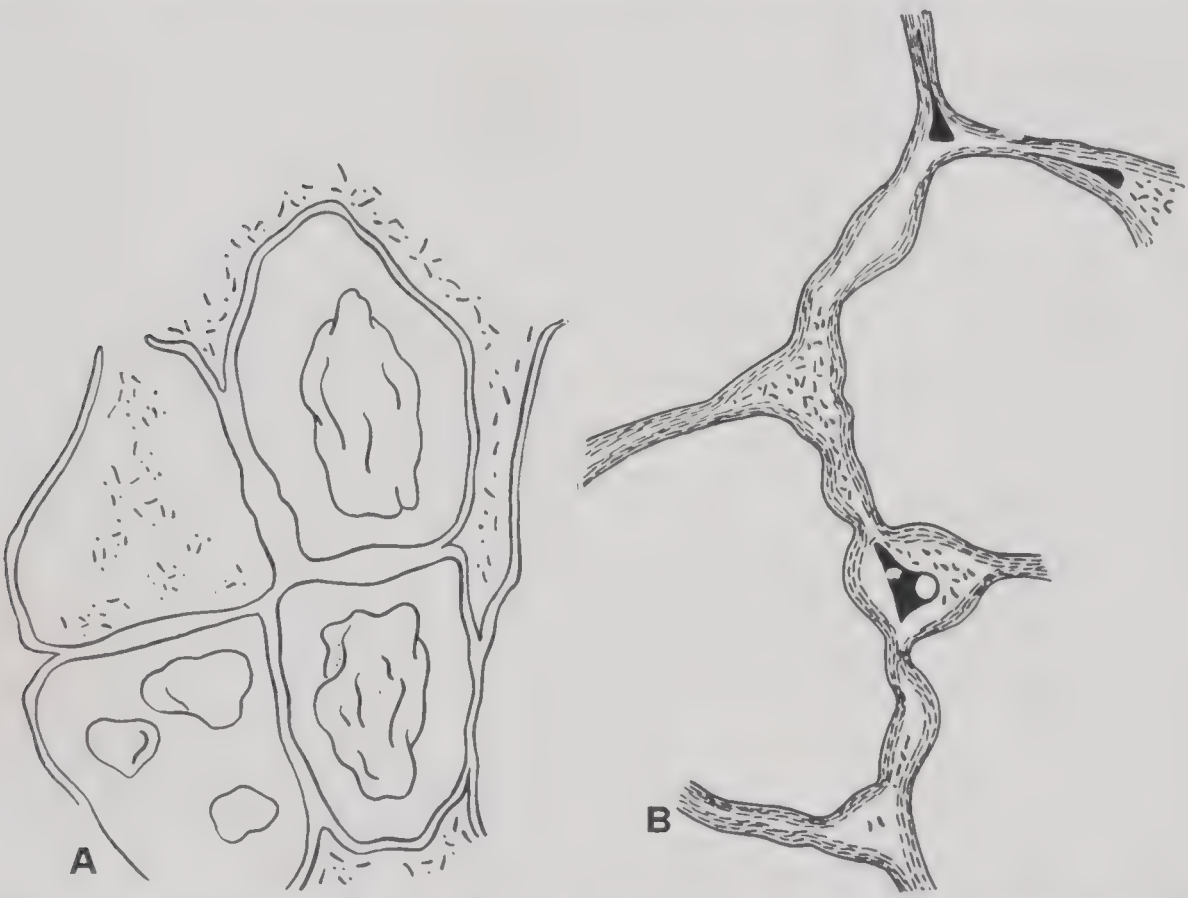

FIG. 15. Relation of soft-rot bacteria to carrot root tissue. *A*, intercellular invasion by the bacteria except for occasional intracellular development when the cellulose walls are injured; *B*, stages in the dissolution of the middle lamella. (*After L. R. Jones.*)

as a source of food for further bacterial growth. Eventually cells in the older decayed tissues become plasmolyzed. The continuation of this process accounts for the watery condition and for the loss of consistency of the decayed tissue. Jones showed clearly that the pathogenic action was actually brought about by the diffusion of the enzyme in advance of the bacteria, while the latter grew upon liquefied lamella and the exosmosed material.

Control. In so far as soft rot of storage vegetables is concerned, the chief means of control have to do with handling of the crop at harvest.

Bruising of plant parts should be avoided. Provision should be made for healing of wounds and for drying of surfaces. Storage should be at the lowest temperature at which the particular product can be expected to retain its culinary quality. With the practice of washing potatoes at harvest before shipment, heavy losses are sometimes suffered in transit because of the ideal conditions for infection by soft-rot bacteria. This may be corrected by running potatoes through a warm-air current before they are sacked (15). For control of potato blackleg, potato refuse piles should be destroyed to prevent them from becoming a source of inoculum.

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FIRE BLIGHT OF PEAR AND APPLE

This disease is very destructive on pear and to a lesser extent on apple. Quite a number of species of the family Rosaceae including some wild

and ornamental species are affected (29). First observed in New York before 1800, the disease has spread throughout much of the United States and Canada. Fire blight is so severe on certain desirable varieties of pear, such as Bartlett, that commercial culture of this fruit has been abandoned in many otherwise favorable areas in the Middle West and East. The pear-growing industry has moved largely to certain regions along the Pacific coast. The disease is least severe in the coastal valleys of that region, but the largest production is still in the inland Sacramento valley of California and in the Rogue River valley of Oregon.

The disease is of historical importance because it is the first one in which a bacterium was shown to be the inciting agent. Beginning in 1878, T. J. Burrill (4), Professor of Botany at the University of Illinois, published a series of papers in which he reported the results of his studies showing the relation of bacteria to the disease. Some 10 years later Arthur (2) repeated and extended the studies, using Koch's recently published poured-plate technique. It was Waite (31) who, a little later (1891), working in the U.S. Department of Agriculture, reported that honeybees, visiting the blossoms of infected branches, became contaminated with the bacteria and transmitted them to other blossoms on the same and other trees. This was one of the first cases of experimental proof of insect transmission of organisms pathogenic on plants. Jones (13) was the first to describe the disease on stone fruits. Fire blight is an important disease in New Zealand and Japan. Although it has been prevalent in Italy since early in this century, and occurs in the Near East and in eastern Europe, it has not as yet become prevalent in western Europe and in the British Isles.

Symptoms. Fire blight appears first on blossoms, sepals, leaves, and twigs. The blossoms turn brown, and the discoloration extends to pedicel and often down the spur. Leaves may be affected at the margins or on the blade, lesions sometimes being checked or sometimes enveloping the whole blade. Dieback extends down twigs and water sprouts, killing the tissue rapidly and turning it to a characteristic brown color (Fig. 16). The dead leaves persist, giving the appearance of having been scorched. Especially in warm humid weather, recently invaded parts may exude amber-colored droplets of exudate made up of bacteria in the gelatinous matrix of plant extract and bacterial by-products. The exudate sometimes spreads to form a thin glistening layer. It dries down to hard beads or films. Under greenhouse conditions Ivanhoff and Keitt (11) observed bacterial exudate in the form of aerial strands up to 45 μ in diameter and several centimeters in length, consisting of bacteria and a cementing substance. While these were not found in nature, they may possibly occur sometimes. They are readily detached and transported by air currents.

When twig blight extends to limb, trunk, or root, localized or diffuse cankers develop, varying in size and shape. As they dry out, they may

become separated from the normal tissue by rifts. They may girdle twigs and branches, and when they affect the base of the trunk, the disease is known as collar blight. It should be recognized, however, that collar blight may be incited by other agencies as well.

FIG. 16. Fire blight of apple. *A*, twig blight; *B*, bacterial exudate on infected stem; *C*, stem canker originating from an infected spur. *(Courtesy of G. W. Keitt.)*

The Causal Organism. *Erwinia amylovora* (Burrill) Winslow *et al.*, 1923.

SYNONYMY:

Micrococcus amylovorus Burrill, 1882.
Bacterium amylovorus (Burrill) Chester, 1897.
Bacillus amylovorus (Burrill) Trevisan, 1899.

The organism is a rod with peritrichous flagella.

Disease Cycle. Blighted twigs and stem cankers form the chief loci of overwintering organisms. The cankers are more consistent sources of primary inoculum in some varieties of the host than in others. The organisms are most active in the living host tissue at the outer edge of the canker, where drops of exudate form on the surface in the spring.

Waite (31) showed that the bacteria may be carried from blossom to blossom by honeybees, and this has been regarded in some regions as the chief means of dissemination. Some investigators have shown that the concentration of the sugar and other constituents of the nectar is important in determining whether the bacteria can multiply when introduced into it (27).

Rain is a major factor in dissemination of the pathogen from holdover inoculum to blossoms, as shown by Gossard and Walton in Ohio (5) and Miller in Wisconsin (18). Miller, in a 3-year study of dissemination on apple, found insects to be of secondary importance. He showed that if the source of inoculum was present in the upper part of the tree, the region of secondary infection brought about by the downward spread of bacteria by spattering rain was cone-shaped, a pattern which would not be explained if bees were the chief means of dissemination. If the holdover cankers were on the lower branches of the tree, the extent of secondary infection was much less. Tullis (30) considered rain dissemination important in Michigan. In some regions water dissemination is apparently not regarded as the most important means of spread, and insect transmission is considered to be more important.

Infection takes place through the openings, which resemble stomata, in the receptacle cups of the blossom, through stomata in sepals and leaves, and through wounds (Fig. 17). The bacteria develop first, apparently in a motile state, in the intercellular spaces of the tissue. As they increase in number, they are embedded in a matrix the nature of which is not known. Haber (6) and Nixon (20) believed that the bacteria migrate in zoogloeal strands, but Miller (18) and Rosen (24) did not agree with this interpretation, while Ivanoff and Riker showed that a similar phenomenon in crown gall was explained by the migration of liquids in intercellular spaces due to negative pressure and capillarity (p. 140). After the exosmosis of water and solutes from the host cells, plasmolysis and relatively rapid death follow. There is evidence of some cell-wall dissolution, and bacteria are found within cells occasionally. The rapid advance of the pathogen is, however, largely through the intercellular spaces. The progress of the disease is more rapid at 24° and 28° than at 16°C. (25).

Varietal Resistance. The trees of some varieties of pear are killed quite rapidly by stem and tree cankers while those of some other varieties are very resistant. Differences in resistance of varieties were recorded as

early as 1817 (7). Apples as a whole are more resistant than pears, but some apples, such as Transcendent crab and Yellow Transparent, blight quite readily. Among the resistant varieties of pear Kieffer is the most widely planted although the fruit is inferior in quality. It is a hybrid between Chinese Sand Pear and Bartlett, the latter being one of the choicest but one of the most susceptible commercial varieties. Under favorable conditions for the disease Kieffer often has severe blight. Richard Peters, developed in Pennsylvania, is another resistant variety. Reimer (21) has conducted the most extensive study of resistance in pears and wild relatives at the Southern Oregon Experiment Station. Most current varieties used in America are members of *Pyrus communis L.*, seedlings of which are used as root stock. Most of these are very susceptible to blight, and in regions such as Oregon where trunk and root blight are severe more resistant root stocks were found in *P. calleryana* Decne. A

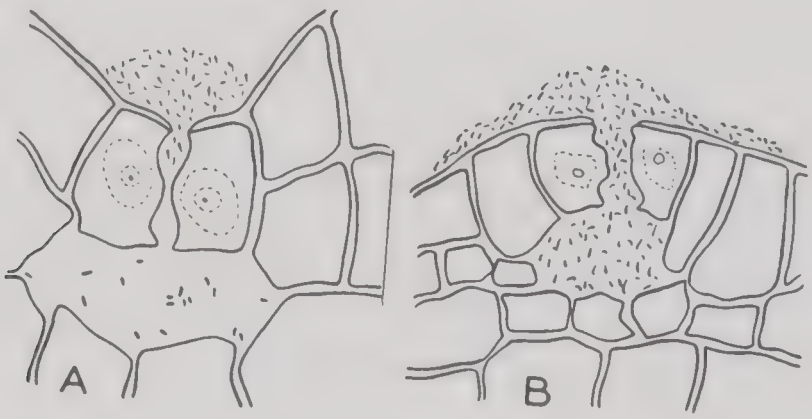

FIG. 17. Stomatal invasion by the fire-blight bacteria. *A*, in nectary; *B*, in sepal. (*After E. M. Hildebrand.*)

few fairly resistant varieties in *P. communis*, such as Old Home, are valuable as trunk and body stocks. Until a resistant variety desirable from the standpoints of root hardiness, productivity, and quality is secured, a temporary solution may be had by a synthetic tree. This is made up of a resistant root stock of *P. calleryana*, a body and trunk of one of the more resistant varieties such as Old Home, and a top of the desirable commercial but susceptible stock such as Bartlett. Grafts are made on several of the resistant branches, and if blight kills one down to the resistant framework, a new graft may be made in place of the one which has been killed. In areas such as that in Oregon where humidity and rainfall are relatively low and where root and trunk blight are the most important phases of the disease, such procedures are practicable. It is doubtful, however, whether the top of such a tree would survive in middle western and eastern areas, where blossom and twig blights are the most serious phases of the disease. Thomas and Ark (28) found resistance to be partially dominant in a cross

between *P. augustifolia* Ait. (susceptible) and *P. gibbsii* var. *yunnanensis* (resistant).

Brooks (3) grouped apple varieties in Wisconsin as follows:

Very susceptible: Hyslop crab, Transcendent crab, Yellow Transparent, McMahon, Fameuse, Wealthy.

Moderately susceptible: Tolman Sweet, Black Ben Davis, Jonathan, Salome.

Moderately resistant: McIntosh, Duchess of Oldenburg, Grimes, Orange Winter, Wolf River, Whitney crab, Stayman Winesap.

Resistant: Ben Davis, Charlemoff, Delicious, Dudley, Northwestern Greening, Tetofsky.

It has been observed by many investigators that the external environment and the type of growth of the various organs have an important bearing upon the current susceptibility of any given tree. Shaw (25, 26) studied the nature of resistance in apple. He found that low soil nutrients, high and low soil temperatures, and high air temperature tended to make plants more resistant, since they tended to favor early cessation of growth. High atmospheric humidity, on the other hand, tended to increase susceptibility. A critical study of intercellular humidity led him to regard this factor as a major one influencing susceptibility. Link and Wilcox (15) and Nightingale (19) studied the carbohydrate-nitrogen ratio of the tissue in relation to susceptibility. They found shoots high in nitrogen and low in carbohydrate very susceptible, while those in a reverse ratio of nitrogen and carbohydrate were very resistant. However, it may well be the succulence produced with high nitrogen, rather than the carbon-nitrogen ratio, which affects the growth of the bacteria in the tissue. Thomas and Ark (28) found tissue high in nitrogen more susceptible in general than the nearest comparable tissue which was lower in nitrogen. Thus an inherently resistant tree may become quite susceptible when humidity is high and high-nitrogen fertilization is practiced, while an inherently susceptible variety may appear fairly resistant when air temperature is high, humidity low, and the tree is in a high-carbohydrate, low-nitrogen condition. Variation between isolates of the organism from several hosts and from various parts of the United States was found by Ark (1).

Control. A generally satisfactory control of this disease on susceptible varieties of pear and apple has not been worked out. The successful culture of very susceptible varieties of pear is limited to areas where the climate is relatively unfavorable to fire blight. Spraying with Bordeaux mixture during the blossom period gives partial control of blossom blight (17, 18). Excision of blighted twigs and small cankers or treatment of cankers with a bactericide reduces primary inoculum. This procedure seems to work well on pears in California, but in Wisconsin it brings about

greater succulence the next year and with it increased susceptibility and eventual pruning near the ground level. The regulation of soil fertilization to reduce the degree of succulence of new growth is advisable. This means caution in the use of readily available nitrogenous fertilizer. Hildebrand and Heinecke (10) in New York found that trees brought to good vigor in an alfalfa cover crop were less subject to blight than those on sod or under clean culture with the application of readily available nitrogen. This was thought to be due to the mechanism whereby nitrogen was liberated by the alfalfa and taken up by the tree. In areas where climatic conditions are favorable to fire blight the extremely susceptible varieties should be avoided. The top-working of resistant varieties of pear has been mentioned in the previous section.

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ANGULAR LEAF SPOT, OR BLACK ARM, OF COTTON

Angular leaf spot was described in Alabama by Atkinson (3) in 1891. Smith (18) was the first to reproduce the disease and to describe the causal organism (1900 1905). It occurs in most if not all cotton-growing regions in the world. It is common throughout the southern states and is of major importance in the Sudan, Uganda, Trinidad, and Turkestan.

Symptoms. The disease appears first on the seed leaves as they come above ground. These leaves take on a water-soaked appearance, turn black, and dry up, often leaving the young green stalk with a blackened tip. Most of such plants die. In less severely affected ones, points on the petioles and stems become water-soaked and enlarge into angular spots about 1 mm. in diameter. The spots often coalesce, and the leaf gradually yellows and drops. Dark lesions occur on the young stems, sometimes causing girdling and death, the phase referred to as black arm. In older

stems the spots may be callused off and leave open cankers. Yellowish bacterial exudate is common on lesions in moist weather. On bolls, gray specks grow to water-soaked circular spots, which coalesce to form irregular, large, brown, sunken areas. Bolls affected when young may drop; if older when affected, they may become distorted and the lint may be discolored.

The Causal Organism. *Xanthomonas malvaccarum* (E. F. Sm.) Dows., 1939.

Synonymy:

Bacterium malvacearum E. F. Sm., 1905.
Bacillus malvacearum (E. F. Sm.) Holland, 1920.
Phytomonas malvacearum (E. F. Sm.) Bergey *et al.*, 1923.

The organism has one to four flagella at each pole.

Disease Cycle. Bacteria gain entrance to the mature seed through the basal end of the chalaza (20). They overwinter in this manner and as contaminants on the surface of the seed or in the lint attached to it. When the seed germinates, the basal cap adheres to the cotyledon and is carried above ground as the plant emerges. Volunteer seedlings are the chief source of primary inoculum when cotton is planted after cotton (11). Soil and meteoric water serve as means of dissemination of the bacteria to new leaves and to the stem. Infection takes place through stomata. The factors in secondary dissemination have received careful study. Faulwetter (8, 9) showed that insects had little importance but that wind-blown rain was the chief disseminating agent in the southeastern states. In semiarid regions, such as Arizona (12), irrigation water is the chief means of secondary spread. Surface water was also found to be the chief means of secondary spread in Uganda (10). In some regions wind-blown infected detached leaves (16) and wind-blown dust (5) may serve as means of dissemination.

Primary infection in the soil appears to develop more readily at the cooler end of the range at which cotton germinates and grows. Relative humidity of 85 per cent or above is essential for abundant infection. Secondary infection is favored by high temperatures, the optimum being about 35°C. (19).

Varietal Resistance. Relatively little has been done in the United States in the development of resistant varieties. All but a few of the current strains of upland cotton are quite susceptible under optimum conditions for the disease (14, 17, 21, 22). Long-lint cottons (*Gossypium barbadense* L.) are very susceptible, a fact which may account in part for the importance of the disease in Arizona.

The most active work on development of resistant varieties has been carried out by Knight and Clouston (13) in the Sudan. They found in

resistant Uganda B31 (an American Upland type of *G. hirsutum* L.) two major dominant factors for resistance (B_1 and B_2) which are cumulative in effect. A third factor B_3 was found in *G. hirsutum* var. *punctatum* J. B. Hutchins., Silow, & Stevens. The long-lint Sakel cotton used in the Sudan is derived from the American Sea Island cotton (*G. barbadense* L.). All strains of this variety are very susceptible. By crossing with Uganda B31 and other resistant varieties various resistant Sakel lines have been secured. Thus this disease is being controlled satisfactorily in some cotton areas by development of resistant varieties.

Control. J. F. Duggar of Alabama recommended in 1911 delinting the seed in concentrated sulfuric acid to control the seed-borne anthracnose organism, *Glomerella gossypii* (Southworth) Edg. Rolfs (15) in South Carolina applied this method successfully for angular leaf spot (1915). Brown and Gibson (6) devised a machine for the purpose (1925). This still remains the most effective means of treatment, although complete elimination of bacteria within the seed is not always accomplished (4, 7). A gravity separation by passing treated seed through water and eliminating floating seed, removes a large share of infected seed (7). Subsequent treatment with 2 per cent Ceresan or New Improved Ceresan dust has additional value.

Since the chief source of primary inoculum other than planted seeds is the volunteer seedlings from seeds overwintering in the ground, early killing of volunteers by cultivation is highly important. This should always occur well before the first watering in irrigated culture. Where suitable resistant varieties are available, they constitute the most effective means of control.

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BACTERIAL BLIGHTS OF BEANS

Blight of common bean (*Phaseolus vulgaris* L.) was first described in the United States by Beach in New York (1892) and by Halsted in New Jersey (1892). The first of the causal organisms was described by E. F. Smith in 1897 (20). The disease which it incites is now referred to as **common bacterial blight**, although it is not necessarily the most common one in any given situation. In 1926, Burkholder (2) described a second bacterial blight of bean, **halo blight**, which has much in common with the originally described disease. In 1930, Burkholder (3) described two other bacterial blights, which have symptoms close to those of common blight and halo blight. These are **fuscous blight** and **bacterial brown spot**. Common blight and halo blight are widespread in the United States and Canada east of the Continental Divide. They occur west of the Divide but are usually of little economic importance. They are widespread in Europe and on other continents where beans are grown under environment favorable for the diseases. Halo blight is at present more widespread and destructive than common blight in the United States and in northern Europe.

Fuscous blight is incited by a variety of the causal organism of common blight. It was found first in Switzerland in 1924. It was not known in the United States until Burkholder reported it in New York in 1944 (4). Bacterial brown spot is incited by an organism which was described first in connection with bacterial blight of lima bean and cowpea. It was shown to be a pathogen of bean by Burkholder (3). It is relatively rare on common bean in the United States. In 1938 its occurrence was reported in Australia (26).

There are several other bacterial diseases of common bean, but most of them are relatively unimportant and have little in common with the bacterial blights. One which does overlap in some degree is wilt. The pathogen, *Corynebacterium flaccumfaciens* (Hedges) Dows, is a vascular parasite, and some of the symptoms are similar to those of the bacterial blights (10).

Symptoms. Common blight is first seen as small translucent water-soaked spots on leaves. The spots arise from stomatal invasion by the bacteria and do not exceed 1 mm. in diameter as a rule. However, the intervening tissue may turn yellow and die to form lesions of dead tissue of various sizes and shapes. Small spots appear also on pods of different ages (Fig. 18). On wax-podded varieties of common bean small slightly sunken spots, brownish to reddish in color, appear and often coalesce. On green pods the spots may be water-soaked at first and turn reddish with age. Water soaking in spots or streaks may appear on petioles and stems and particularly on the pulvinus at the juncture of branch or petiole and main stem. As the disease progresses, brownish discoloration of the vascular system may become evident, and brownish surface cankers may form on the stem in the vicinity of the first node. The top-heavy podded plant may break over at this point, giving rise to a phase of the disease known as joint rot. The seeds show various degrees of shriveling and discoloration depending upon the extent of infection. **Halo blight** has most of the symptoms of common blight. One striking point of difference is that around individual spots on the leaf a chlorotic zone or halo up to $\frac{1}{2}$ in. in diameter forms promptly. Exudate may form on pod and stem lesions with both diseases; the exudate of halo blight is cream- or silver-colored; that of common blight is yellowish.

Fuscous blight ordinarily cannot be distinguished from common blight, except by isolation and study of the causal organism. The lesions on the stem are more inclined to split the stem than in common blight (3). **Brown-spot blight** appears as water-soaked spots which remain very small and become reddish brown in color. When the organism becomes systemic, sunken longitudinal lesions occur on the stem. Pale brown halos, about $\frac{1}{2}$ in. in diameter, sometimes appear around spots on

the pod. In **wilt**, spotting of leaves and pods does not occur. The leaflets wilt and eventually die. A water-soaked lesion extends along the suture of the pod. Stem cankers similar to those in common and halo blights may occur. Infected seed is often shriveled and discolored. The signs on the seed are not distinct enough, however, to facilitate elimination of the pathogen by hand picking.

Common and halo blights are important transit diseases of snap beans (15, 18).

FIG. 18. Common bacterial blight of bean.

The Causal Organisms.

Common blight: *Xanthomonas phaseoli* (E. F. Sm.) Dows., 1939.

SYNONYMY:

Bacillus phaseoli E. F. Sm., 1897.
Pseudomonas phaseoli E. F. Sm., 1901.
Bacterium phaseoli E. F. Sm., 1905.
Phytomonas phaseoli (E. F. Sm.) Bergey *et al.*, 1923.

The organism has a single polar flagellum.

Fuscous blight: *Xanthomonas phaseoli* var. *fuscans* (Burk.) Stan. & Burk., 1942.

SYNONYMY:

Phytomonas phaseoli var. *fuscans* Burk., 1930.

The chief difference between this variety and *X. phaseoli* is in the brown water-soluble pigment produced by *fuscans* in most media.

Halo blight: *Pseudomonas phaseolicola* (Burk.) Dows., 1943.

SYNONYMY:

Phytomonas medicaginis var. *phaseolicola* Burk., 1926.
Bacterium puerariae Hedges, 1927.
Bacterium medicaginis var. *phaseolicola* (Burk.) Link & Hull, 1927.
Pseudomonas medicaginis var. *phaseolicola* (Burk.) Stapp & Kotte., 1929.
Phytomonas puerariae (Hedges) Bergey *et al.*, 1930.

The organism has a single polar flagellum.

Brown-spot blight: *Pseudomonas syringae* van Hall, 1902.

SYNONYMY:[1]

Bacterium syringae E. F. Sm., 1905.
Bacterium viridifaciens Tisdale & Williamson, 1923.
Pseudomonas viridifaciens Tisdale & Williamson, 1923.
Pseudomonas vignae Gardner & Kendrick, 1923.
Phytomonas vignae (Gardner & Kendrick) Bergey *et al.*, 1923.
Phytomonas syringae Bergey *et al.*, 1930.

The organism has one or two polar flagella.

Bacterial wilt: *Corynebacterium flaccumfaciens* (Hedges) Dows., 1942.

SYNONYMY:

Bacterium flaccumfaciens Hedges, 1922.
Phytomonas flaccumfaciens (Hedges) Bergey *et al.*, 1923.
Pseudomonas flaccumfaciens (Hedges) Stevens, 1925.
Phytomonas flaccumfaciens (Hedges) Stapp, 1928.

The organism has a single polar flagellum.

Disease Cycles. The common and halo-blight organisms overwinter in diseased vines (11, 27) and in the seed. When seed is infected, the bacteria develop in the seed coat of the germinating seed and contaminate the surface of the expanded cotyledon, penetrating through rifts in the cuticle and progressing intercellularly until the vascular system is reached (Fig. 19). They may then proceed as vascular pathogens throughout the large xylem tubes of the plant. Both leaf lesions and stem cankers may arise from systemic infection (28). Primary local infection may also take place through stomata by inoculum arising from infected cotyledons or plant debris. Continued spread of secondary inoculum from primary lesions is carried out by wind-borne rain, dust, implements, and man and animals. The blight phase may develop rapidly under favorable conditions. Common blight is favored by relatively high air temperatures, while halo blight thrives under relatively cool conditions (7). Thus they may become epi-

[1] For a complete list of synonyms see "Bergey's Manual," 6th ed., pp. 119–120.

demic in different regions and at different times of the season. Pods are infected locally or by systemic vascular invasion. Seeds are infected by surface contamination from the pod wall or by systemic invasion through the funiculus.

The bacteria migrate through the intercellular spaces of parenchyma tissue, dissolving the middle lamella slightly in advance. The affected cells collapse and are invaded. This breakdown of the tissue leads to formation of lysigenous cavities. The xylem cells are also invaded, and

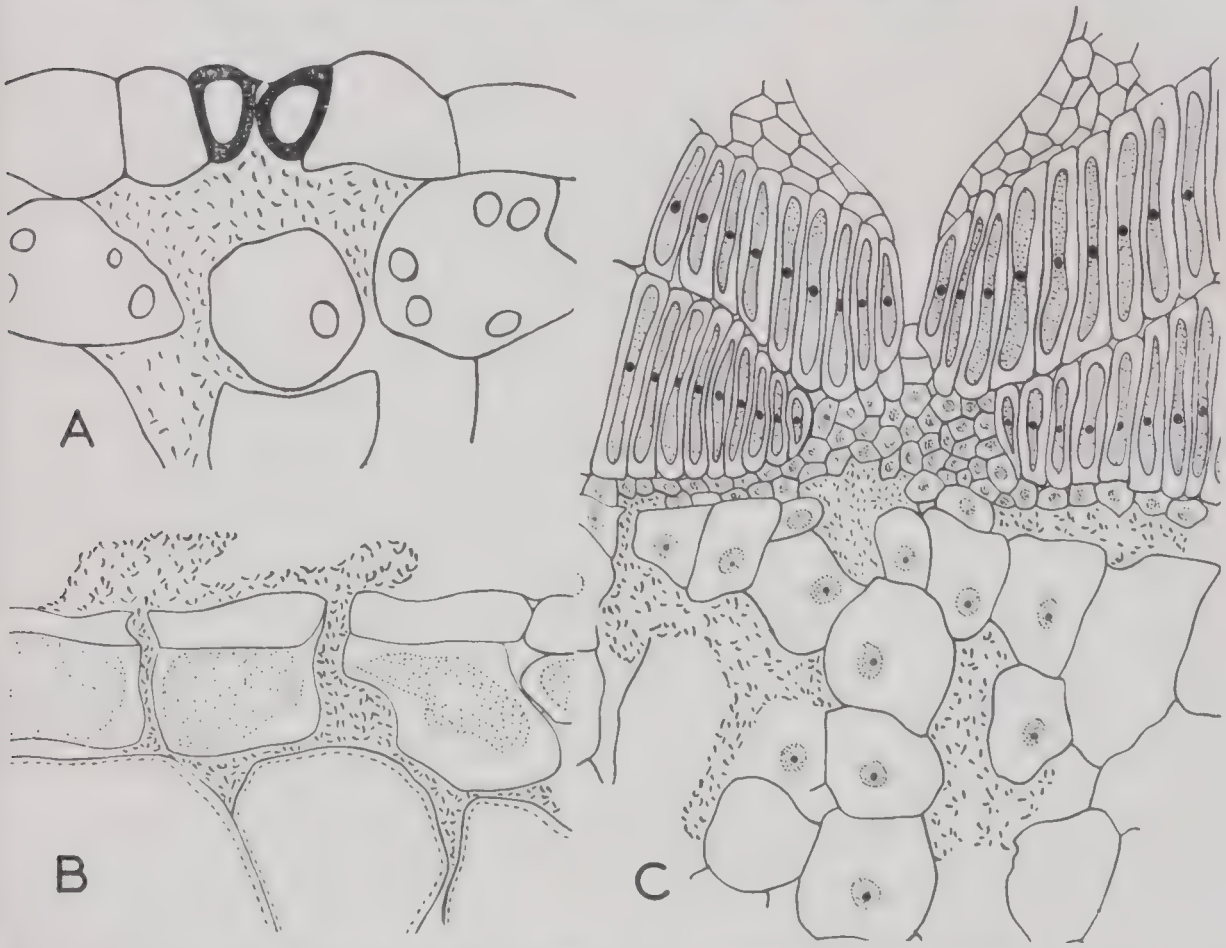

FIG. 19. Common bacterial blight of bean. *A*, invasion through stoma; *B*, invasion through rift in the cuticle of the cotyledon; *C*, invasion of the seed through the tissue of the funiculus. (*After Zaumeyer.*)

the bacteria may advance systemically in the larger xylem elements. When the invaders multiply to large masses in xylem cells, they either rupture or dissolve the cell walls and reenter the parenchyma tissues. They may emerge through stomata on the stem and enter other stomata on the same stem after dissemination. These two organisms are, therefore, both parenchyma and vascular invaders (28).

The wilt organism is confined more strictly to the vascular system. It enters the plant through the cotyledon from the infected seed coat or through leaf and stem wounds. It does not invade stomata, and secondary

infection during the growing season is known to occur only through wounds. In all three diseases infection of the seed occurs systemically through the funiculus or through the micropyle, and the organisms are thus well protected in the seed. Furthermore, infected seeds serve as the chief means of distribution and perpetuation of the pathogens (28). The relation of *P. syringae* to lima-bean tissue is described by Beach (1).

Host Range. The bacterial-blight organisms and the wilt organism affect various other hosts within the legume family. The range of susceptibility of some of the common legumes as reported by Burkholder (3) is given in Table 1.

Table 1. Host Range of Bean Bacterial-blight and -wilt Organisms

Legume	X. phaseoli	X. phaseoli var. fuscans	P. phaseoli-cola	P. syringae	C. flaccum-faciens
Common bean	+[a]	+	+	+	+
Small lima, or Sieva, bean	+	+	+	+	+
Scarlet runner bean	+	+	+	0	+
Hyacinth bean	+	0	0	+	+
Adzuki bean	0		0		+
Cowpea	0	0	0	+	+
Yard-long bean	0	0	0	+	+
Soybean	0	0	0	+[c]	+
Kudzu vine	0	0	+[b]	+	0
Broad bean	0	0	0	+	0
Field pea	0	0	0	0	0
White sweet clover	0	0	0	0	0
Alfalfa	0		0		
White-flowered lupine	+	0	0	0	0

[a] + = inoculation tests positive; 0 = inoculation tests negative.
[b] Reported positive by Hedges (10) but not by Burkholder (3).
[c] Infection on pods only.

Control. Crop rotation of about 3 years is essential and is sufficient to dispose of overwintering organisms. Sanitation with bean straw should be practiced rigidly. The use of clean seed is desirable, but such seed is often difficult to secure. Both common and halo blight occur in most seed-growing areas, although some of the irrigated regions in the Rocky Mountain states and in California have climates less favorable to the diseases than others.

Varieties vary in susceptibility to common and halo blight, but a given variety may react differently to each disease pathogen. None are completely immune, but some are sufficiently tolerant to justify their use in preference to others (12, 13). Lists of resistant and susceptible varieties

have been published for common and halo blight by several investigators (5, 6, 8, 9, 21–24, 27).

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BLACK ROT OF CRUCIFERS

This disease was first recorded in Kentucky in 1891 (3). The bacterial pathogen was described from rutabaga in Iowa in 1895 by Pammel (6). At about this time it became destructive in numerous cabbage-growing areas in the United States and was studied intensively in Wisconsin by Russell (8) and in this and other states by Smith (9 11). The pathogen was one of the first of the bacteria shown to be seed-borne; this was brought out by Harding *et al.* (4) in 1904 in studies of the disease on cabbage seed plants on Long Island. Owing undoubtedly to seed transmission of the pathogen, the disease has been reported to occur, often destructively, in many parts of the world. It occurs on many cultivated and wild crucifers. Cabbage, cauliflower, Chinese cabbage, turnip, and rutabaga are among the cultivated hosts commonly affected severely. Other hosts include Brussels sprouts, broccoli, kohlrabi, collard, radish, rape, charlock, black mustard, and Indian mustard.

Symptoms. In most instances infection occurs through water pores at the margins of leaves, and the first signs of disease often appear at this point. Occasional lesions start at wounds caused by chewing insects. In either case the tissue turns yellow, and the chlorosis progresses toward the center of the leaf, usually in a V-shaped area with the base of the V toward the midrib. In the chlorotic tissue the veins and veinlets become dark in color, and when the lesion is held before a source of bright light, the network of black venation stands out prominently. The vessels of the larger veins and midrib may turn black in advance of the lesion while in the midrib the tissue turns brown, dehydrates, and becomes brittle. Vascular discoloration extends to the main stem and proceeds upward and downward.

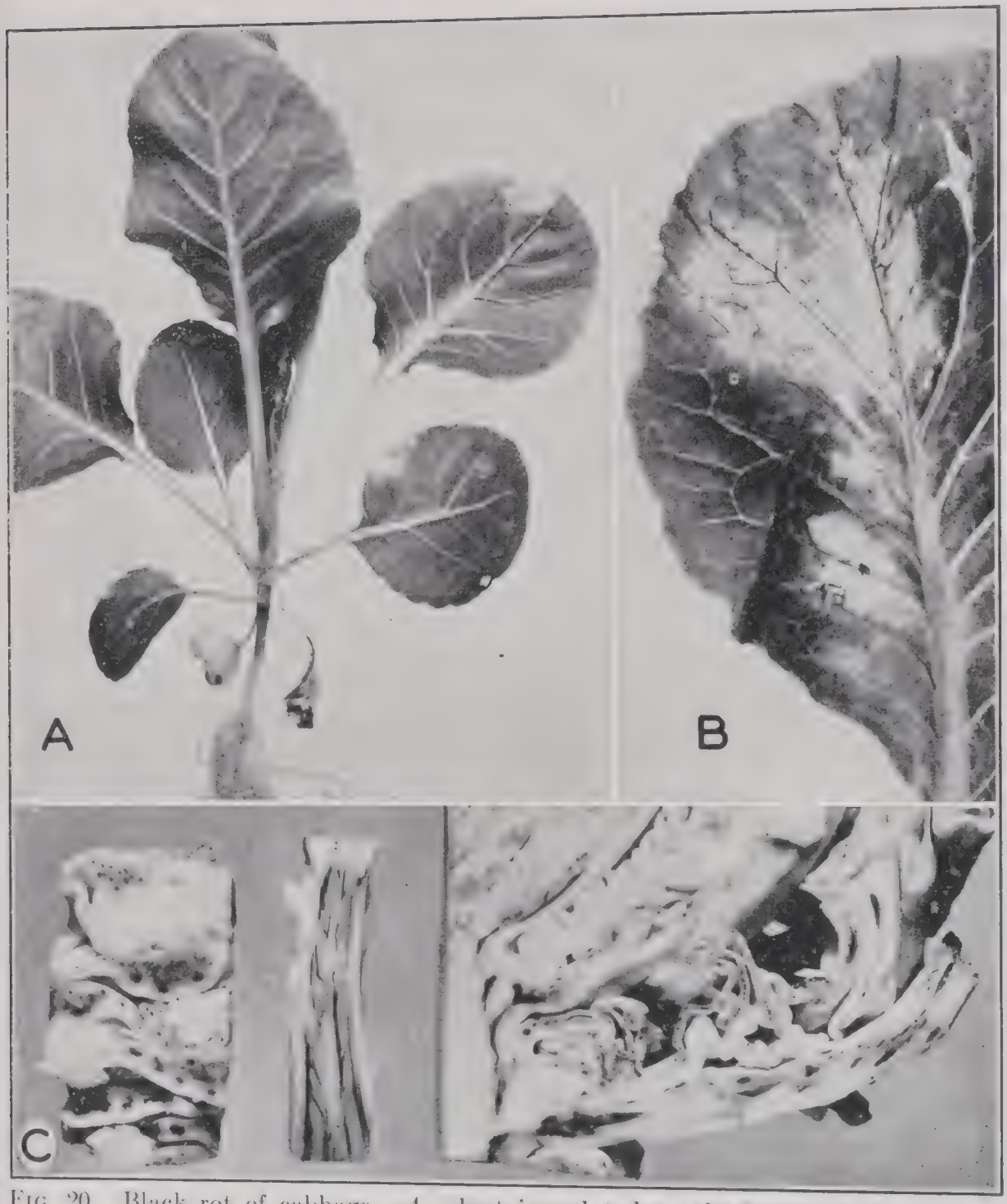

FIG. 20. Black rot of cabbage. *A*, plant inoculated on the lowest leaf, which is now dead. Following systemic invasion, secondary symptoms appeared at the leaf margins. Such lesions are indistinguishable from those which arise from primary invasion of the hydathodes at the leaf margin. *B*, advanced systemic lesion in older leaf. Note blackening of veins and veinlets against the chlorotic parenchyma tissue. *C*, blackened veins in stem, midrib, and head leaves.

Lesions may occur on one or both of the cotyledons, which turn yellow and drop prematurely but usually not until the organism has reached the stem through the cotyledonary node. The upward advance of vascular symptoms may proceed into upper leaves, and chlorotic lesions from systemic invasion may appear anywhere on the leaf (Fig. 20). Leaves so invaded may be

stunted unilaterally. Abscission layers at the bases of affected leaves may develop prematurely, and early defoliation then occurs. Cabbage and cauliflower heads are invaded and discolored. In turnip and rutabaga, the fleshy root is invaded from the leaves, the vascular bundles showing black discoloration and later internal breakdown. The soft-rot bacteria (p. 101) commonly follow into fleshy storage tissue and destruction continues as soft rot.

The Causal Organism. *Xanthomonas campestris* (Pam.) Dows., 1939.

Synonymy:

Bacillus campestris Pam., 1895.
Pseudomonas campestris (Pam.) E. F. Sm., 1897.
Bacterium campestris (Pam.) Chester, 1897.
Bacterium campestre (Pam.) E. F. Sm., 1911.
Phytomonas campestris (Pam.) Bergey *et al.*, 1923.

The organism is a rod with a single polar flagellum.

Disease Cycle. The organism overwinters in diseased plant refuse and in seed. In either case the bacteria usually enter first through stomata at the marginal sinus of the cotyledon lobe, to which the infected seed coat is often attached and carried above ground during growth. From the substomatal chamber the bacteria progress intercellularly until they reach and invade the extremities of spiral vessels (2). In the latter they progress systematically throughout the plant. In the leaves, stomata are not invaded, but infection occurs through water pores at the leaf margins, and from these loci they advance through the intercellular spaces to the spiral vessels (Fig. 21). It is not uncommon for infection to occur on cabbage and cauliflower in the seedbed, when cotyledons and one or two lower leaves become infected and drop off prematurely. With unfavorable conditions for further dissemination the external signs of disease may disappear for several weeks, the bacteria progressing slowly within the vascular tissue of the stem. Visible systemic lesions may appear some weeks after transplanting in the upper leaves. Bacteria which reach the surface of such lesions and are spread by spattering rain are the chief means of local dissemination. By systemic invasion of seed stalks, the bacteria may reach the siliques (seed pods) internally. Seeds may thus be contaminated on the surface, but to some extent invasion of the seed coat takes place through the vascular system in the funiculus. Mildly affected seeds are capable of germination the following season, and from their seed coats bacteria gain access to the cotyledon. After a protracted dormant period in the early part of the growing season, the disease commonly increases rapidly under favorable conditions of temperature and moisture, and epidemic development ensues. Ofttimes an epidemic is traceable to a given lot

of infected seed or to a seedbed which became infested early from inoculum in refuse or in seed. Insect transmission by larvae of the cabbage worm is known but is not important. The chief means of local dissemination are wind-blown rain, cultivating implements, and transplants. Widespread distribution is by seed and transplants.

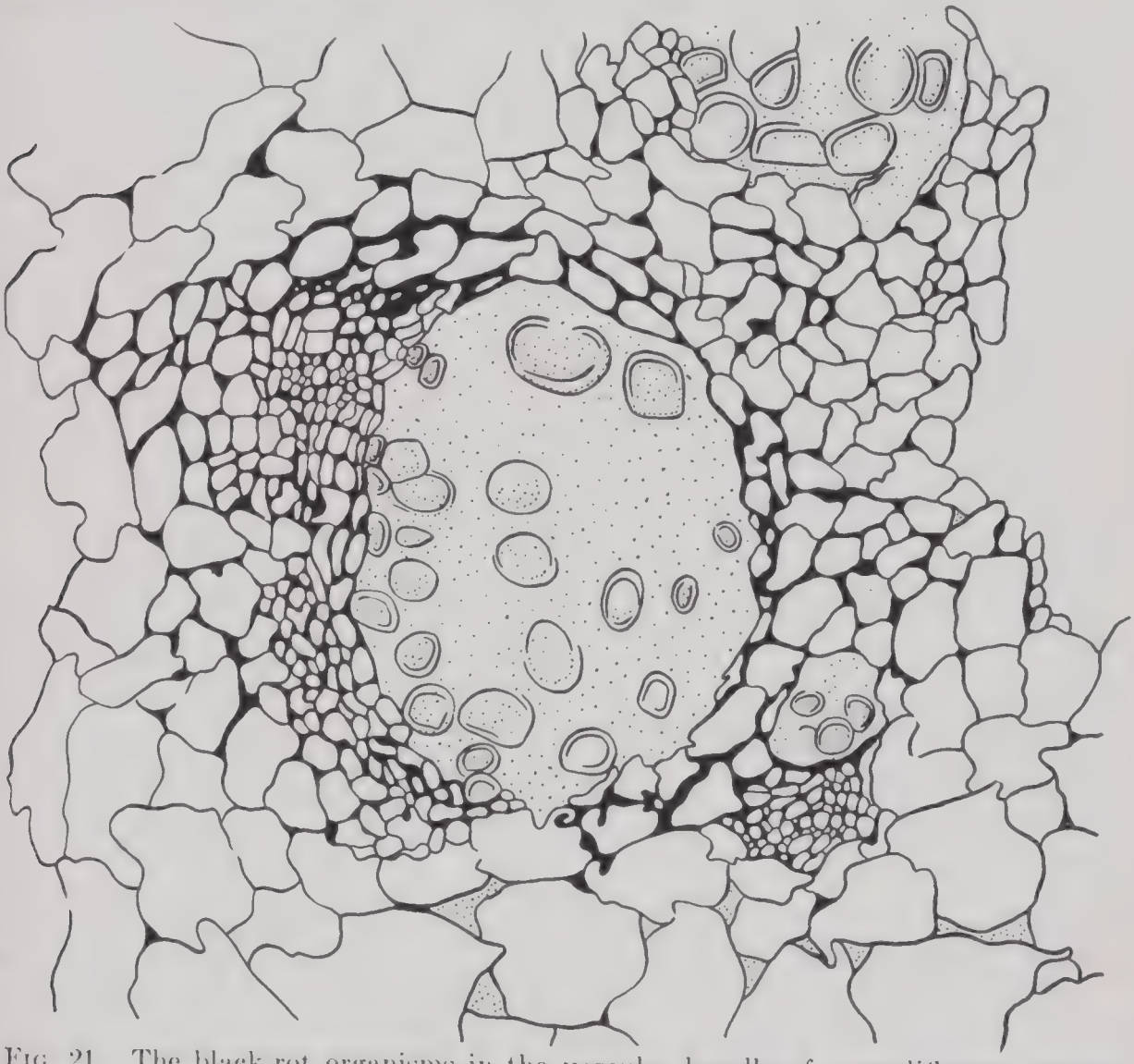

FIG. 21. The black-rot organisms in the vascular bundle of a cauliflower petiole. Note disintegration of xylem, without damage to phloem, followed by intercellular invasion of parenchyma. (*After E. F. Smith.*)

In regions where rainfall is very low during the period when seedlings are being grown, the absence of this important disseminating factor precludes development of the disease. In the region around Puget Sound and in many other Pacific coast areas such climate occurs. Black rot is little known and practically absent in these localities. Since most United States grown seed of cabbage, cauliflower, turnip, and rutabaga is produced in these areas, the seed from such sources is practically always free from the black-rot organism. This is an outstanding example of producing

seed free from a seed-borne pathogen by growing the seed crop in an environment which prevents infection.

Control. Seedbed rotation is important. Three years' absence of susceptible crucifers is necessary. Seed should be disinfected by treatment in water at 50°C. for 30 min., unless it is certain that the seed is free from the black-rot and blackleg organisms.

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SOUTHERN BACTERIAL WILT

Southern bacterial wilt affects a great many hosts (12), and it has become known by many common names, including Granville wilt of tobacco and brown rot of potato. In the United States it is most prevalent in the southeastern states, particularly on tomato, tobacco, potato, eggplant, pepper, and peanut. It is a common disease of tomato in home gardens in that region. Until a resistant variety was developed, it was severe on the tobacco crops in North Carolina, where in the sandy soils of Granville County much of the good tobacco land was at one time abandoned for this crop, because of heavy infestation by the wilt pathogen. It is a serious disease of potato, eggplant, and pepper in the coastal areas of North Carolina, in the Hastings area of northern Florida, and elsewhere in the southeastern states. It is common in many tropical countries. In Indonesia it is a major disease of tobacco and potato; in the Philippine Islands it is destructive on tobacco, tomato, and eggplant; in many tropical regions it

affects banana. The first record of the disease appears to be on tomato in Italy in 1882. In addition to the above-mentioned countries it has been reported from Japan, India, Ceylon, North Australia, and South Africa. The first monographic treatment was by Smith (11) in 1896.

Symptoms. On tomato, the lower leaves may droop before wilting occurs. The vascular system becomes brown, and development of adventitious roots along the stem may be enhanced. In potato the leaflets take on a bronze color, shrivel, and die, and leaves may wilt. The vascular system of stem and tuber turns brown. Whitish bacterial exudate oozes from the vascular system of cut stems and cut tubers. In damp soil the bacterial mass may exude from the eyes of the tubers, to which soil adheres when the crop is dug. When young tobacco seedlings are affected, the wilt develops rapidly. The older plants are stunted with less permanent wilting of the leaves, and the vascular system is discolored. Older leaves of eggplant wilt first and young leaves later, but the disease is less severe on this host than on tomato and tobacco. Peanut foliage loses its green color, wilts suddenly, and turns dull black.

The Causal Organism. *Pseudomonas solanacearum* E. F. Sm., 1914.

Synonymy:

Bacillus solanacearum E. F. Sm., 1896.
Bacillus nicotianae Uyeda, 1904.
Bacillus sesami Malkoff, 1906.
Pseudomonas sesami Malkoff, 1906.
Bacillus musae Rorer, 1911.
Bacterium solanacearum E. F. Sm., 1914.
Bacillus musarium Zeman, 1921.
Erwinia nicotianae (Uyeda) Bergey *et al.*, 1923.
Phytomonas solanaceara (E. F. Sm.) Bergey *et al.*, 1923.
Phytomonas ricini Archibald, 1927.

The organism has one polar flagellum.

Disease Cycle. The pathogen is a soil inhabitant, persisting for long periods of years in some soils. It invades the plant through wounds, usually below ground. Actual penetration has not been studied carefully, and it is not known whether the bacteria invade through rifts in the root cortex by secondary roots, or otherwise. The organisms migrate first in the large xylem vessels, and in succulent stems they progress into the intercellular spaces of the cortex and pith, causing lysigenous cavities (Fig. 22). In potato tubers the organism may produce an internal decay emanating from the vascular bundles and causing a breakdown in the bud tissue of the eye.

The physiological response of the host varies with the degree of infection.

Invasion of a single lateral bundle in a tomato petiole causes drooping (epinasty) while invasion of all bundles causes wilt. When vessels of the tomato are invaded, there is a tendency for adventitious roots to develop in the tissue just outside of the invaded bundle. The wilting is thought to be due to a gradual mechanical blocking of the xylem vessels, but it may well be that further study will reveal that toxic materials produced by the pathogen supplement the physical factors in producing symptoms.

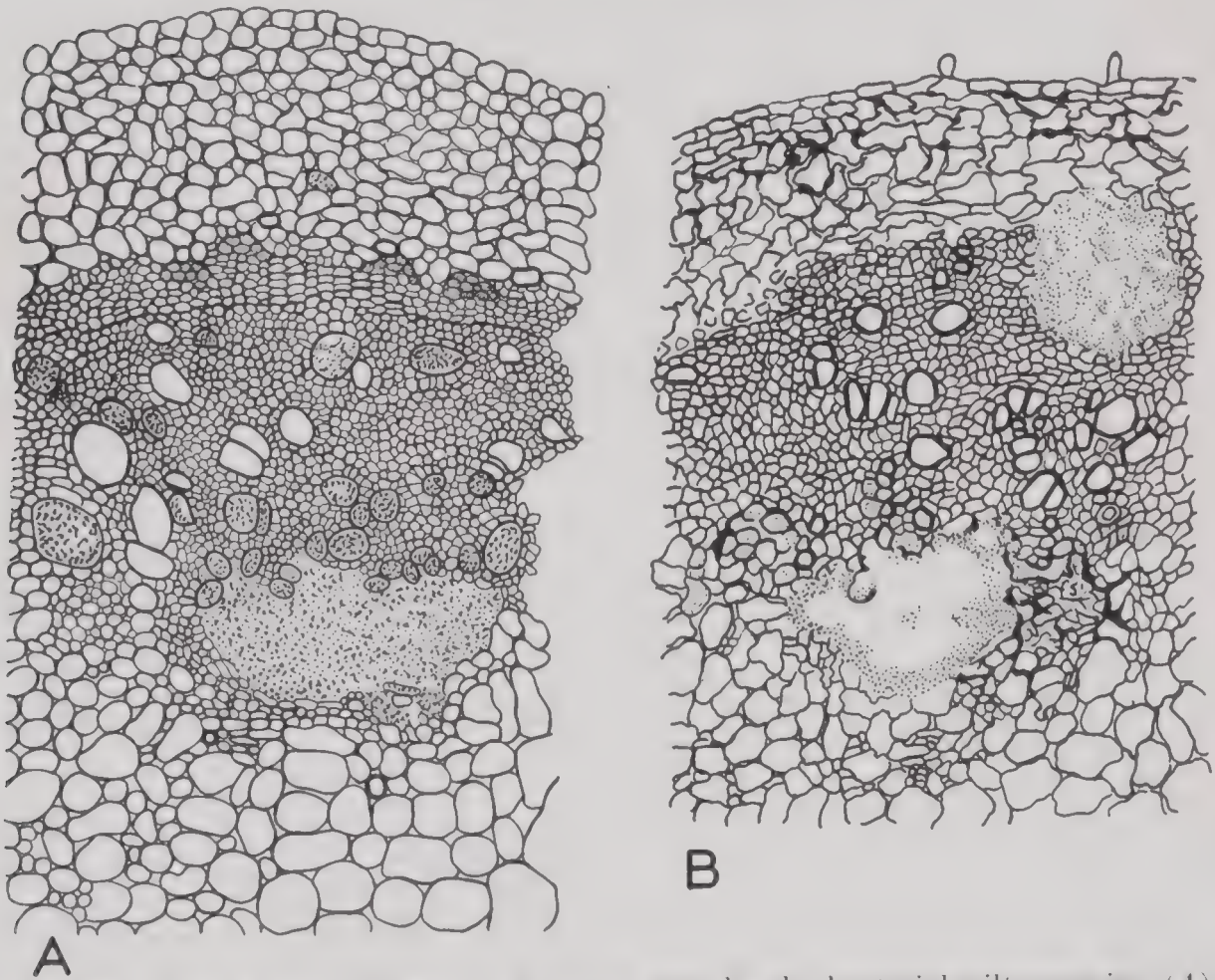

FIG. 22. Comparison of invasion of tomato stem by the bacterial-wilt organism (*A*) and by the bacterial-canker organism (*B*). Note that the former is confined to the xylem while the latter is concentrated in the inner and outer phloem elements. (*After E. F. Smith.*)

The organism becomes established in southern soils, but the disease seldom occurs north of Virginia, although it is probably introduced into northern soils repeatedly on transplants of tomato and pepper. Potato tubers also are a means of distribution.

Relatively high soil moisture and soil temperature favor the disease. The severity of infection was increased in Indonesia by superphosphate and decreased by nitrogenous fertilizers. Smith (13) found that nitrogenous fertilizers, particularly urea, could be used as a partial control on

tobacco in some North Carolina soils, but it was necessary to use 1,000 lb.-per acre and to grow a crop of corn between the application and the tobacco crop in order to maintain quality of the latter. Eddins (4) found that increase of the hydrogen-ion concentration of the soil to pH 4 for a summer period, followed by a fall application of lime to bring the reaction to about pH 5, reduced the organisms in the soil sufficiently to be commercially practical.

Control. When the pathogen becomes established in soil, satisfactory control is difficult. Eddins's sulfur-lime treatment, mentioned above, is feasible when the crop is valuable enough to warrant the expense, and it is necessarily limited to certain soil types. In tobacco lands some control is accomplished by rotation (2) and fertilization (13). In the Granville area of North Carolina, a very successful resistant variety has been developed which has brought tobacco growing back to a profitable level in this area (14). Some control through resistant varieties of vegetables has been accomplished. At the Puerto Rico Experiment Station a resistant strain of tomato, LJX-7, was developed by crossing Louisiana Pink with a native resistant type (9). Resistant eggplants have been reported in the Philippine Islands (10), Ceylon (8), and Puerto Rico (9). A resistant peanut is reported in Indonesia (16). Green Mountain and Katahdin varieties of potato were more resistant in northern Florida than other varieties grown there (3).

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BACTERIAL WILT OF CUCURBITS

Bacterial wilt is a common and often destructive disease on muskmelon and cucumber. Squash and pumpkin are susceptible but are not affected as severely. A number of cucurbits, including watermelon, have been infected artificially. The disease occurs throughout the United States and Canada east of the Rocky Mountains; it is more common in the northern states than in the south. Wilt has been reported in Europe, South Africa, and Japan.

Symptoms. The first signs of wilt appear usually on individual leaves, which become flaccid at the edges in sunny weather (Fig. 23). They turn dull green in color as the disease progresses; more leaves wilt, and eventually an entire branch is affected. The wilting then becomes permanent, and the leaves and vines shrivel and die. Occasionally an exudate on fruits is visible. When wilted stems are cross-sectioned, the viscid sticky bacterial matrix exudes from the bundles and may be drawn out in strands an inch or more. This feature is used as a means of diagnosis.

The Causal Organism. *Erwinia tracheiphila* (E. F. Sm.) Holland, 1923.

Synonymy:
Bacillus tracheiphilus E. F. Sm., 1895.
Bacterium tracheiphilum (E. F. Sm.) Chester, 1897.

The organism has four to eight peritrichous flagella, and capsules are formed.

Disease Cycle. The bacteria overwinter in the bodies of adult cucumber beetles, of which there are two species, the striped beetle (*Acalymma vittata* Fabricius) and the spotted beetle (*A. duodecimpunctata* Olivier).

Primary infection is produced when beetles feed upon young leaves or cotyledons. This is the only means of natural infection known. This pathogen is one of the unusual cases in which the organism is completely dependent upon the insect for its survival. After infection, the bacteria invade and progress within the tracheal vessels. The capsular material of the organisms provides a viscid matrix in the vessels and is believed to

FIG. 23. Bacterial wilt of cucumber. Primary invasion has occurred through beetle wounds in four of the lower leaves, which have wilted. As systemic vascular invasion proceeds from these loci, the whole vine will soon wilt rather suddenly.

cause wilt by means of mechanical plugging of the xylem (6, 12). Whether or not other more diffusible toxic by-products of the bacteria have a part in pathogenesis is not known, but such a possibility is to be considered. Secondary spread from primarily infected vines is carried on by beetles. The thermal death point of this organism is distinctly lower than that of other plant pathogens. Smith (12) suggested that its sensitivity to heat might be a reason why it occurred less commonly below the latitude of Virginia.

Varietal Resistance. There is a considerable difference between cucumber varieties in their susceptibility to wilt, but none are very highly resistant. Doolittle (4) observed that certain European varieties tested were more susceptible than American varieties. There is some indication that varieties may be improved in resistance by breeding (5). As yet no highly resistant varieties of cucumber or melon have been released.

Control. Since the pathogen is entirely dependent upon the beetle for its perpetuation and dissemination, control of wilt involves control of the insect. It is not enough to reduce insect damage to a commercially acceptable degree, since insects will still feed on the plants in sufficient numbers to cause an epidemic. It is essential, therefore, to apply insecticides early, to forestall the colonization of overwintering adults, and to continue applications frequently. There are good indications that a fungicide applied with the insecticide enhances the degree of control. Since cucurbits are sensitive to DDT, it should be avoided or used with caution as an insecticide on these crops.

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RING ROT OF POTATO

This disease was found in Germany early in the present century, where it was described in 1913 by Spieckermann. It was reported in eastern Canada in 1931 and in Maine in 1932. It appeared in rapid succession in various regions of the United States and Canada. By 1940 it had been found in 37 states, and in some instances it had caused extremely heavy losses. It had not been reported in Great Britain up to 1949. The pathogen has been found in nature only on potato. It has been shown experimentally to be infectious on tomato, eggplant, and some other related species of the Solanaceae (6, 9).

Symptoms. In the field the disease appears first as a diffuse and progressive pale yellow chlorosis of leaflets, which may flag under some conditions. Marginal necrosis appears next and progresses along with chlorosis. The entire plant shows stunting and may wilt in some cases. The chief distinctive feature, however, is chlorosis and marginal burning. Examination of the interior of the stems of plants showing advanced symptoms reveals browning of the vascular elements. By squeezing, a milky viscous bacterial exudate may be forced out of the cut stem (Fig. 24). In tubers the disease sometimes may not be seen before harvest, symptoms appearing later in storage. The first sign in the tuber is a vascular discoloration, which is light yellow, becoming brown with age. Later the bacterial ooze may be evident, and it may be intensified by squeezing the tissue. As the bacterial exudate becomes more copious, breakdown of the tissue of the vascular ring to form macroscopic lysigenous cavities follows. Dehydration then occurs, and the advanced lesions may take on a dry, powdery appearance or they may become soft and mushy with the invasion by soft-rot bacteria. The vascular cavities may extend to pith and cortex and in the latter may result in canker lesions on the surface of the tuber. Ring rot should be compared with brown rot (southern bacterial wilt), with which it has some overlapping symptoms (4).

The Causal Organism. *Corynebacterium sepedonicum* (Spieck. & Kotth.) Skapt. & Burkh., 1939.

Synonymy:

Bacterium sepedonicum Spieck. & Kotth., 1914.
Aplanobacter sepedonicum (Spieck.) E. F. Sm., 1920.
Phytomonas sepedonica (Spieck.) Magrou, 1937.
Corynebacterium sepedonicum (Spieck.) Dows., 1939.

The organism is nonmotile.

Disease Cycle. The rapid spread of this disease in North America was associated with dissemination of seed tubers from special growing areas in northern states and Canada. The organism lives from season to season chiefly in tubers. It will subsist also in the dried slime on crates, bins, harvesting and grading machinery, and sacks. There is no evidence that it persists in the soil. However, volunteer plants from surviving tubers in

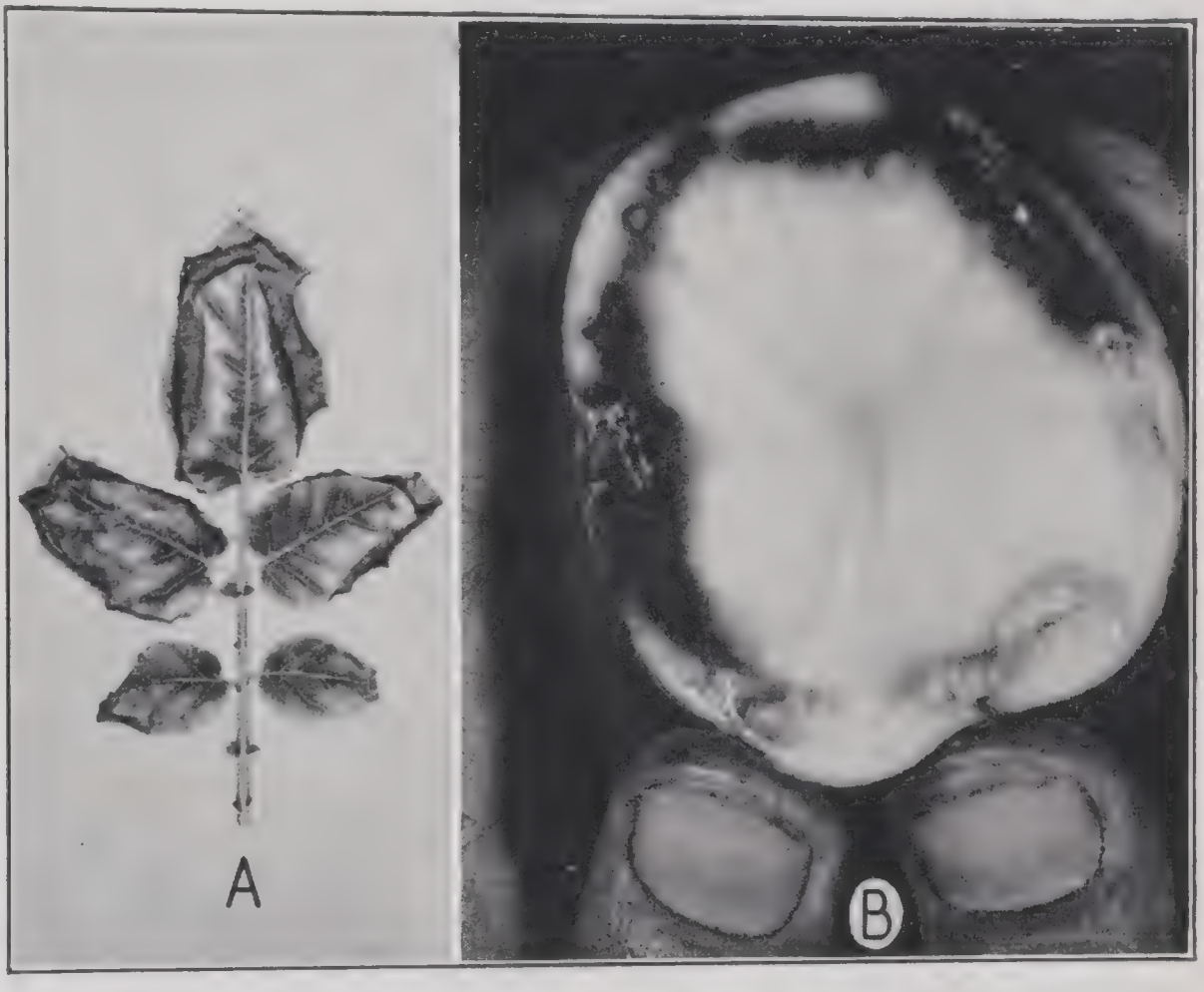

FIG. 24. Ring rot of potato. *A*, chlorosis and marginal necrosis of leaflets of systemically infected plant; *B*, the first sign in the tuber is the creamy exudate which may be forced from the vascular ring of a cross-sectioned specimen, as seen in the lower portion of the photograph. Later invasion of parenchyma leads to dark discoloration and often a powdery disintegration. (*Courtesy of R. H. Larson.*)

the soil may be a source of primary inoculum (2). The initial invasion is through wounds. There is no evidence of invasion through stomata, lenticels, or water pores. Since the bacterium is resistant to drying in the slime mass, contaminated machinery and cutting knives are the chief source of primary inoculum, which enters through cut seed pieces and through harvest and handling bruises. There is some evidence of spread in the field by irrigation water and from hill to hill through the root system. The disease becomes established in the large vessels of the vascular system

of stem and tuber and progresses in this channel systemically (7). Eventually the bacterial mass progresses into xylem parenchyma and contiguous tissue, where lysigenous cavities are formed. The bacteria may remain for a long time in tubers without producing visible symptoms. It is possible for a tuber to carry the germs, internally, and produce symptomless plants and symptomless tubers although all three stages are infected. This is especially true when growth of the host is under continuously cool environment. Larson and Walker (10) showed that no top symptoms appeared in plants from infected tubers grown at an air temperature of 16°C., while stunting was severe at 24°C. Sherf (14) secured maximum infection of stolons and tubers in plants growing at a soil temperature of 18°.

Experimental transmission through tomato seed has been demonstrated (9).

Control. Since the bacterial-ring-rot organism is distributed on seed tubers, rigid tightening of certification standards followed its appearance in the United States. In most states no visibly infected plants or tubers are permitted in seed stock to be certified. Since final field or warehouse diagnosis is not always possible, supplementary laboratory procedures are sometimes necessary (5).

When a grower once has ring-rot–infected stock on his property, thorough disinfection of all warehouses, bins, sorting equipment, and cultivation machinery becomes necessary because of the extreme infectiousness of the pathogen and its dissemination by equipment. Used bags should be discarded or thoroughly disinfected. For bins, crates, and bags copper sulfate (25 lb. per 100 gal. of water) is best. For machinery formaldehyde (1–10) or BK (4,000 to 5,000 p.p.m. of chlorine) is recommended. Following such sanitary precautions, only foundation or certified seed stock should be used for planting. All other potato stocks must be kept off the premises.

Since the organism is transmitted from tuber to tuber by the cutting knife, one infected tuber in a lot may lead to a general infection of seed stock by this means. Use of rotary knives which run continuously through 1–500 mercuric chloride has been urged by many investigators. Frequent renewal of chemical disinfectants is necessary to assure favorable results. Continuously boiling water is as effective as chemicals if it is kept at boiling temperature. Another device is a stationary double-edged knife over which 1–500 mercuric chloride flows continually. Picker planters facilitate greater spread from seed piece to seed piece than do assist-feed planters (11).

Some progress has been made in the development of resistant strains. One new resistant variety, Teton, has been introduced (12). It is well to point out that this is not immune and is successful because it produces

satisfactorily in spite of infection. It still remains a possible carrier of the pathogen and thus is a potential source of contamination where susceptible varieties are being grown.

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BACTERIAL CANKER OF TOMATO

The disease was first described in 1909 by Erwin F. Smith on tomatoes growing in a greenhouse at Grand Rapids, Michigan. He first called it Grand Rapids disease but later named it bacterial canker. It was found in

a few northern states in the following 18 years but did not assume major importance until 1926 when it caused considerable damage on outdoor tomatoes in western New York. In the years immediately following, it became a major disease in field-grown tomatoes throughout the canning belt from New Jersey to California and in several southern states. It has caused losses in many other countries including Australia, New Zealand, South Africa, Kenya, Morocco, Germany, Russia, England, and Canada. The disease has been found in nature only upon tomato, except for one instance reported from southern California (2) in which stem cankers were found on perennial nightshade in a tomato field. The isolate from nightshade was infectious on tomato and on nightshade, but an isolate from tomato was not infectious on nightshade. Ark (1) produced infection by artificial inoculation on tree tomato (*Cyphomandra betacea* Sendt.) on red-currant tomato (*Lycopersicon pimpinellifolium* Mill.), on *Solanum nigrum* var. *guineense* L., and on *Nicotiana glutinosa* L.

Symptoms. Temporary, and later permanent, wilting of leaflets appears at the extremities of plants of any size. The next signs are light streaks beginning at the juncture of leaf petiole and stem, extending down the internode and up the petiole (Fig. 25). As these streaks become darker, the epidermal tissue may rift, and a longitudinal canker-like opening may appear in stem, petiole, or midrib. There may be a stunting of the plant, or occasionally young plants may appear to recover as they grow older. Leaves may be affected unilaterally, the leaflets on one side of the rachis being stunted or wilted while those on the opposite side appear healthy. When the stem of a diseased plant is cut lengthwise, a creamy white, yellow, or brown line follows the phloem. Cavities in the older diseased tissue extend into the pith or into the cortex and to the outer surface. The discolored vascular system can be traced to the fruit, where it becomes brown and, ramifying to the outer surface, may cause brown blister-like exterior lesions. The discoloration may also extend to the placentae and the seeds.

The symptoms described above appear as the result of systemic infection by the pathogen. Secondary spread from canker lesions is common. Invasion takes place through stomata on leaves, cotyledons, and stems, small light-colored spots forming which are not often conspicuous in nature. Secondary infection appears on green fruits as water-soaked spots which soon become surrounded with a white halo. The spots remain less than 3 mm. in diameter exclusive of the halo. The presence of a halo has been emphasized by Bryan (5) as a means of distinction between the canker spot and that of bacterial spot incited by another organism, *Xanthomonas vesicatorium* (Doidge) Dows, which is reported not to have a halo. This is not entirely correct, since the latter disease commonly does have a halo

surrounding the fruit lesion in the early stages, and at this time the two diseases are indistinguishable on green fruits. In bacterial spot the halos disappear as the lesion becomes older.

FIG. 25. Bacterial canker of tomato. *A*, wilt of the terminal leaf and development of a stem canker, beginning at a leaf node and proceeding downward; *B*, local lesion on the green fruit consisting of a scabby spot surrounded by a pale-yellow to whitish halo; *C*, bacterial spot, sometimes confused with the canker spot, since in the early stages the spot may also have a halo. The latter disappears as the scabby lesion develops.

The Causal Organism. *Corynebacterium michiganense* (E. F. Sm.) Jensen, 1934.

SYNONYMY:

Bacterium michiganense E. F. Sm., 1910.

Pseudomonas michiganense (E. F. Sm.) Stevens, 1913.

Aplanobacter michiganense E. F. Sm., 1914.
Phytomonas michiganense (E. F. Sm.) Bergey *et al.*, 1923.

The organism is nonmotile.

Disease Cycle. The organism lives over from season to season within the seed coat. It may also be carried on the surface of seeds. Blood (3) discovered that when the fruit pulp is allowed to ferment at room temperature for 72 hr. or more, the bacteria on the surface and within the seed coat are, as a rule, completely destroyed. Fermentation of pulp in the commercial extraction of tomato seed has long been a standard procedure, and this fact accounts in a large measure for the minor importance of the disease until 1926. About the latter date, cyclonic machines for extraction of tomato juice were introduced in canning factories. Since it was possible to save both juice and seed by this process, the need for fermentation in seed extraction was removed. However, the omission of fermentation allowed perpetuation of the canker organism, and seed became the means of rapid and widespread dissemination. As a result, the disease was elevated from a minor to a major role in the United States, and it began to appear in many other countries, possibly for the same reason.

The seed coat is commonly attached to the cotyledon and carried above ground after germination. Invasion of the cotyledon through stomata is commonly the locus of primary infection. Signs of the disease may then disappear, as in black rot of crucifers, while the organism grows slowly in the vascular system. Reappearance of symptoms may come after seedlings are transplanted. In any case they usually show as wilting of leaflets and later as open cankers. Local dissemination is from open cankers and is carried out chiefly by spattering rain. Seed becomes infected internally by systemic invasion of the fruits. Widespread distribution of the pathogen is by seed and by transplants.

The organism becomes established early in the phloem (Fig. 22). It is the only known vascular bacterial organism which migrates through the plant primarily in the phloem. Eventually it invades the cortex and causes lysigenous cavities, which may break open at the surface to cause cankers.

The disease is influenced by various soil and air factors (7). It may develop at air and soil temperature as low as 16° and as high as 36°. The optimum soil and air temperatures for disease development are about 28°C. This is also about the best temperature for growth of the tomato. However, if plants are grown at this temperature or slightly below it before they are infected, they resist the progress of the disease more than plants predisposed at higher or at lower temperatures. Walker and Kendrick (8) showed that after infection the rate of disease development at the optimum

temperature was enhanced considerably by increasing the concentration of the nutrient solution up to a certain level.

Control. Bryan (5) did not succeed in ridding infected seed of the organism by chemical or by hot-water treatment. The best method is fermentation of the seed and pulp at room temperature for 72 hr. before extraction. Blood (4) showed that in the process of extraction the hydrogen-ion concentration of the juice rose considerably, and he surmised that high acidity was responsible for eradication of the bacteria. He devised a soak in acetic acid, which is about as effective as fermentation if it is applied to freshly extracted seed. In addition to fermentation much reliance has been placed in recent years on inspection and certification of seed fields.

There is little evidence of overwintering of the organism in crop refuse. As a precautionary measure for this and other tomato pathogens, sterilization or rotation of seedbeds and rotation of the main field are advised.

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CROWN GALL, HAIRY ROOT, AND CANE GALL

In the bacterial diseases discussed in the previous pages the major effect on the host plant is necrotic and or hypoplastic. Crown gall is representative of a group of diseases in which the major effects on the host are hyperplasia and hypertrophy. This disease affects a considerable number of host plants in nature, and a much wider range of plants has been shown to

be susceptible by experimental inoculation. Crown gall is important on apple, peach, apricot, almond, grape, rose, and cane fruits.

In 1897, Cavara, in Italy, first isolated a bacterial organism from galls on stems of grape and reproduced the disease by inoculation. Numerous other investigators worked with the disease in Europe and America between 1892 and 1904 without successfully isolating an inciting agency. Erwin F. Smith and C. O. Townsend took up a study of the disease in 1904. The pathogenicity of the causal organism was demonstrated by 1906, and the organism was described in 1907.

In 1930, Riker *et al.* (16) distinguished hairy root from crown gall and described the causal organism of the former. It has a more limited host range than the crown-gall organism. It was reported to be pathogenic on apple, rose, honeysuckle, sugar beet, bean, and Paris daisy, but very slightly so or not at all on tomato and tobacco.

Cane gall of brambles was distinguished from crown gall by Banfield (2) in 1934. Hildebrand (11) described the pathogen as a distinct species in 1940. It is confined in its pathogenicity to members of the genus *Rubus*.

Crown gall has been extensively studied because of the opportunity to examine basic causes of pathological growth. The extensive considerations bearing on the cancer problem are too voluminous for discussion here. Reviews by Riker *et al.* (19, 21) should be consulted.

Symptoms. Crown gall consists in overgrowths varying from the size of a pea or smaller to 50 lb. or more. Galls occur commonly on the subterranean roots of trees or shrubs (Fig. 26). They may also appear on crowns, stems, or leaves of woody and herbaceous plants. The overgrowth may be relatively soft and thus is readily disintegrated by secondary organisms; it may consist of a corky exterior and a hard woody interior.

Hairy root, or woolly knot, commonly occurs on grafted nursery apple trees. The distinctive symptoms are excessive production of small roots, sometimes from hard swellings which often occur at the graft unions of piece-root-grafted apple trees.

Cane gall appears as small protuberances or elongate ridges on the fruiting canes of *Rubus* spp., somewhat before the middle of the growing season. These increase in size and may completely cover sections of the surface, especially in the lower portion of the cane. The gall tissue is whitish at first, turning brown with age. As the season advances, severely affected canes split open, the leaves wither, and the berries dry up.

Wound overgrowths, nonparasitic in nature, are confused commonly with crown gall since they may occur along with the latter. Such overgrowths often occur on apple trees at the margin of root and scion, particularly when an imperfect union has been made.

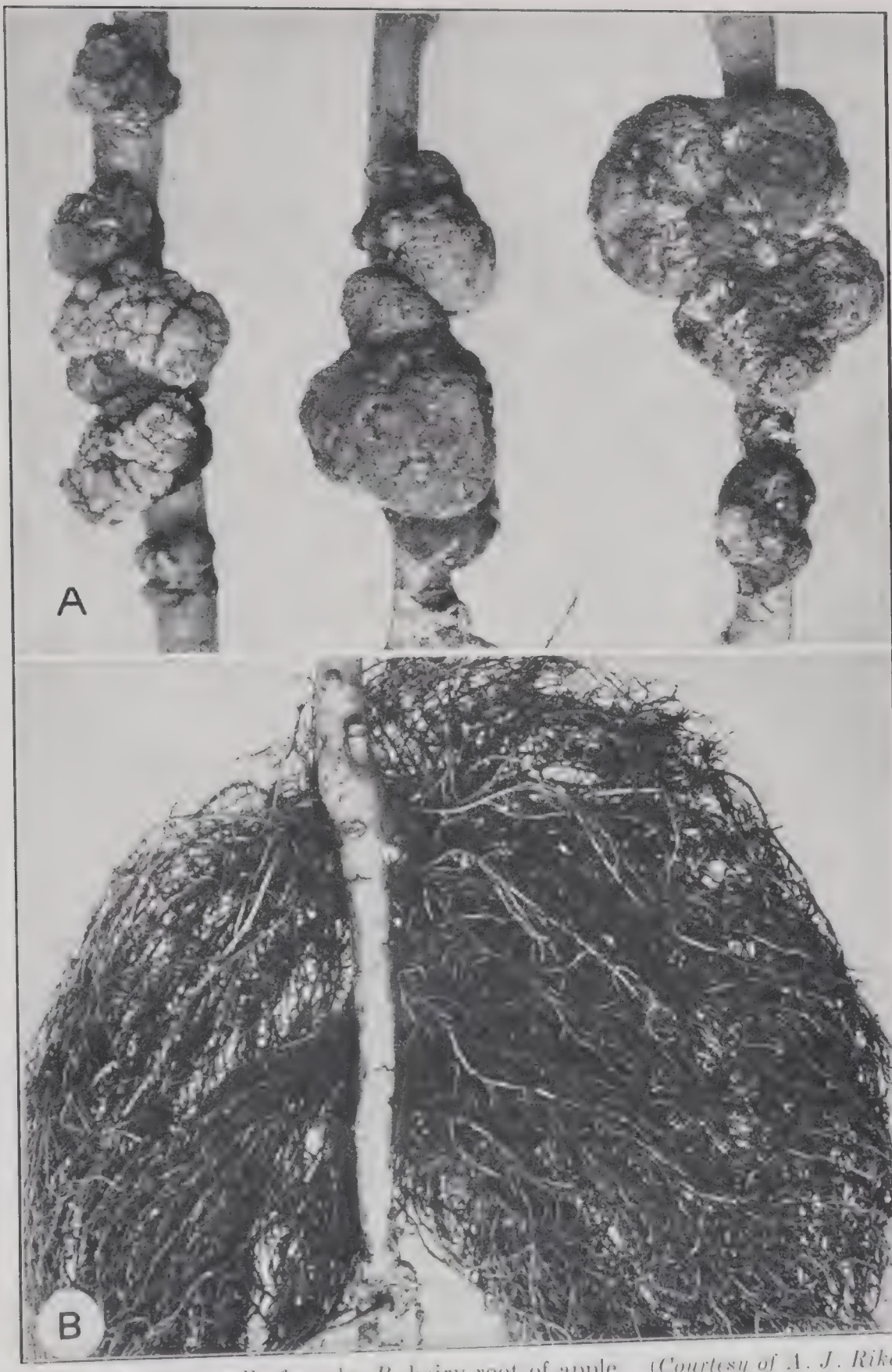

FIG. 26. *A*, crown gall of apple; *B*, hairy root of apple. *(Courtesy of A. J. Riker.)*

Mixtures of crown gall, hairy root, and wound overgrowths are commonly found in the nursery.

The Causal Organisms.

Crown-gall organism: *Agrobacterium tumefaciens* (E. F. Sm. & Towns.) Conn, 1942.

SYNONYMY:

Bacterium tumefaciens E. F. Sm. & Towns., 1907.
Pseudomonas tumefaciens (E. F. Sm. & Towns.) Stevens, 1913.
Bacillus tumefaciens (E. F. Sm. & Towns.) Holland, 1920.
Phytomonas tumefaciens (E. F. Sm. & Towns.) Bergey *et al.*, 1923.

The organism has one to four polar flagella.

Hairy-root organism: *Agrobacterium rhizogenes* (Riker *et al.*) Conn, 1942.

SYNONYMY:

Bacterium rhizogenes Riker *et al.*, 1930.
Phytomonas rhizogenes Riker *et al.*, 1930.
Pseudomonas rhizogenes Riker *et al.*, 1930.

A. rhizogenes differs from *A. tumefaciens* in the following ways: (*a*) it is unable to utilize such simple nitrogenous compounds as potassium nitrate; (*b*) it absorbs congo red and bromothymol blue slightly and aniline blue not at all; (*c*) it is unable to grow on sodium selenite agar; and (*d*) it is able to form acid from various sugars.

Cane-gall organism: *Agrobacterium rubi* (Hildebrand) Starr & Weiss, 1943.

SYNONYMY:

Phytomonas rubi Hildebrand, 1940.
Bacterium rubi Hildebrand, 1940.
Pseudomonas rubi Hildebrand, 1940.

A. rubi differs from *A. tumefaciens* in that it does not utilize nitrates and grows much more slowly on ordinary media. It differs from both *A. tumefaciens* and *A. rhizogenes* in that it does not utilize asparagine as a sole source of carbon and nitrogen.

Disease Cycle. The organisms are successful soil inhabitants. They are distributed widely on living planting stock. Infection takes place entirely, so far as is now known, through one or another type of wound. Subterranean chewing insects are important in facilitating crown-gall infection. Hildebrand (12) found that, in general, the deeper the wound, the better the chances of infection and the larger the gall.

Smith (25) in an early publication described the bacteria as intracellular. However, Riker (14) and Robinson and Walkden (22) found the organisms to be intercellular like other gall-forming bacteria. Bacteria may be carried a short distance from the wound by liquid which fills the intercellular spaces, but usually no farther than inert material would be transported by negative pressure and capillarity.

Rapid cell division takes place around the point where bacteria are present in the intercellular spaces. Hyperplasia and hypertrophy of cells follow. An old gall consists of a mass of such tissue and often elements which resemble scalariform cells of the vascular system.

In 1912, Smith reported secondary tumors, which were connected by strands of tumor tissue, and interpreted his findings as comparable to metastases in animal cancer. This view has been doubted by others. Riker (14) and Robinson and Walkden (22) showed that if the crown-gall bacteria were inserted in wounds near the growing tip of a plant like Paris daisy or sunflower, they might follow the released intercellular liquid through a number of nodes. As the growing tissue elongated rapidly, they would later be found to be distributed in several disconnected loci, at each of which a gall developed. Thus Riker found in one case with sunflower that secondary galls appeared 13 nodes and 49 cm. distant from the point of inoculation. Most workers since Smith agree that tumor strands often fail to connect primary and secondary galls. Braun (4) (1941) claimed the development of sterile secondary tumors and tumor strands in sunflower and secured sterile transmissible tumor tissue from secondary tumors, but it is possible that the bacterium originally present may have died.

In studies on crown gall of tomato Riker (15) found the organism to grow on culture medium from about 10 to 34°C. with an optimum at about 22°. The host grew best at from 22 up to 30° soil temperature. When stems were inoculated below the soil line with crown-gall bacteria, gall development was best at 22°, meager at 28 to 30°, and nil above 30°. Gall development in this case was greatest at the optimum for the pathogen and somewhat below the optimum for the host.

Although most bacterial overgrowths in apple trees in the nursery occur at the graft union, Riker *et al.* (18) found that new infections occurred in wounds during the second, third, and fourth seasons after the graft was made. Infection by the hairy-root organism subsequent to the healing of the union seemed to be correlated with the occurrence of warm weather, with the activity of soil insects, and with rapid growth of the tree. The incubation period for both crown gall and hairy root was relatively long in the spring and fall and relatively short in the summer.

Extensive physiological and biochemical studies have been made with

the organism and upon the fundamental factors underlying atypical growth in the pathological tissues. These are reviewed by Riker *et al.* (19, 21).

E. F. Smith and Quirk (30) described a species of *Begonia* immune to the crown-gall organism. C. O. Smith (24) found a wide range of resistant and susceptible species and varieties within the genus *Prunus*. Some of these have value as root stocks. Wormald (32) has described four physiologic races of the crown-gall organism on the basis of host reaction. The gall-inducing bacteria were compared by Riker *et al.* (21).

Control. The chief approach to control of crown gall, hairy root, and cane gall is through the production of clean nursery stock. Obviously little can be done after the orchard has been set. Inspection and certification is required in all states before removal (see chap. 15), and this to a large extent keeps these diseases in abeyance.

For control in the nursery, certain definite procedures are necessary. Proper disposal of infected stock is paramount. Clean areas where root-chewing insects are not prevalent should be selected for the nursery. Well-fitted grafts wrapped with antiseptic tape, together with sanitary practices in grafting and storage, must be had to ensure a minimum of infection and to reduce the amount of confusion with wound overgrowths. Antiseptic solutions should never be applied to antiseptic tape wrappers, since they unduly delay or prevent suitable decay of the cloth and girdling of the plant may follow.

Ark (1) has reported effective eradication of galls on almond with elgetol (20 parts in 80 parts of methyl alcohol) painted on the stem galls and an inch-wide zone of surrounding bark. Brown and Boyle (7) have shown that soft galls of *Bryophyllum* disintegrated when penicillin was applied to them. They suggested this as a possible means of control.

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COMMON SCAB OF POTATO

There are two scab diseases of potato which are distinguished in popular terminology as common scab and powdery scab. The latter is described in Chap. 5. Common scab has been known for more than 100 years in both Europe and America. The causal relation of the inciting organism was first shown by Thaxter at the Connecticut Agricultural Experimental Station in 1890. The greatest loss from scab is brought about by the blemish to the appearance of the tuber and the consequent reduction in

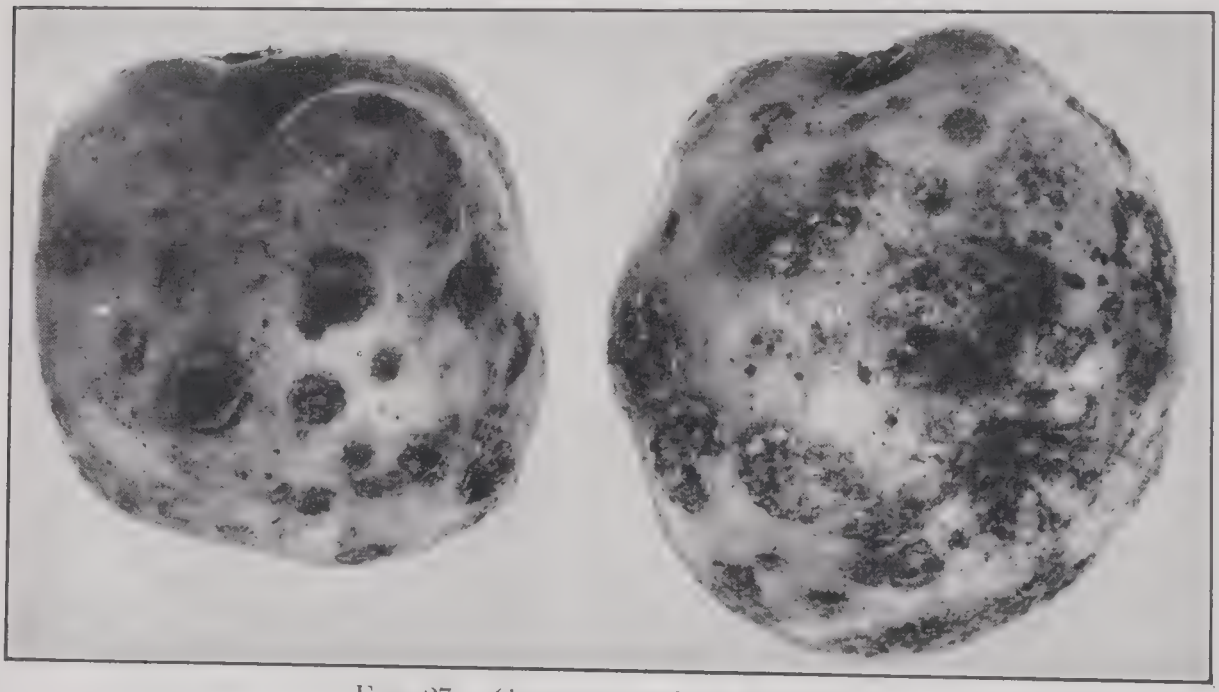

Fig. 27. Common scab of potato.

commercial grade. When lesions are deeply pitted, the waste in peeling is increased. Predisposition of the tuber to chewing insects adds to the loss. The disease is world-wide in occurrence. In highly acid soils it is relatively scarce, while in soil with moderate hydrogen-ion concentration, and particularly in muck soils, it is most prevalent and most important.

Table beet, sugar beet, and radish are common economic hosts.

Symptoms. Shallow and deep scab are two phases commonly referred to in popular and scientific literature. Shallow scab consists in a superficial roughened area, sometimes raised above, and often slightly below, the plane of the healthy skin. It consists of corky tissue which arises from abnormal proliferation of the cells of the periderm of the tuber, resulting from invasion by the pathogen (Fig. 27). The lesions vary widely in size and shape and usually are slightly darker than, but not greatly different in color from, the

healthy skin. This symptom may be such that it causes a diffuse russet, or it may grade into the deep phase.

Deep scab, or pitted scab, consists of lesions which are 1 to 3 or more mm. deep. They may be darker than shallow lesions. The tissue around the interior of the pits is corky, as in shallow scab. Opinions differ as to the nature and origin of deep-scab lesions. One view is that certain physiological races of the organism incite this type of lesions while other races incite shallow scab. Another view is that deep scab is the result of combined action of the scab organism and of chewing insects attracted to the incipient lesions. Relatively inconspicuous lesions may be found on stolons and on subterranean stems. Lesions on roots of other plants in special cultures are described by Hooker (16).

The Causal Organism. *Streptomyces scabies* (Thaxt.) Waksman & Henrici, 1948.

Synonymy:
Oospora scabies Thaxt., 1890.
Actinomyces scabies (Thaxt.) Güssow, 1914.

There are two prevalent points of view concerning the systematic position of the group to which the scab organism belongs. One of these views is exemplified by the conclusions of Drechsler (11):

> Owing to the absence of any well defined bacterial characteristics, . . . the view that *Actinomyces* represents a transition between the Hyphomycetes and Schizomycetes, as well as the phylogenetic corollary based upon it, may safely be abandoned. If mere size is to be regarded as important, it would appear to be equally profitable to look for bacterial affinities in some ascomycetous and sphaeropsidaceous forms, the hyphae of which are similarly very minute. . . . There seems to be no adequate reason why the genus should not be classed in an unqualified manner with the Hyphomycetes, as a mucedineous group with tendencies toward an erect Isarioid habit.

Drechsler came to the above opinion after a thorough study of the morphological characteristics of this group. The opposite view is taken by others who believe that the actinomycetes are closely related to the higher bacteria, in which rudimentary mycelium occurs. In the sixth edition of "Bergey's Manual" the forms which produce conidia in chains on aerial hyphae are placed in the genus *Streptomyces*. The potato-scab organism and the incitant of soil rot of sweet potato, *S. ipomoea* (Person & Martin) Waksman & Henrici, are the only species in the order which are recognized as plant pathogens.

Aerial mycelium in pure culture consists of prostrate branched threads. Sporogenous hyphae are spiral in form (Fig. 28). Spores are produced by

the formation of septa at intervals along the hypha, which contract to form narrow isthmuses between the cells. When the spores in the chain mature, final cleavage takes place. The spores are roughly cylindrical (0.6 to 0.7 by 1.0 to 2.0μ) and hyaline. They germinate by means of one or two germ tubes.

Disease Cycle. The pathogen may subsist indefinitely in favorable soil. It is distributed chiefly on infected seed tubers and in wind- and water-borne soil. It may survive passage through the digestive tract of animals and become distributed with manure.

The young tuber is most susceptible to infection. The pathogen enters chiefly through lenticels but also through stomata, wounds, or directly through the cuticle when the latter is very scanty. It progresses intercellularly at first, apparently digesting the middle lamella. The invasion

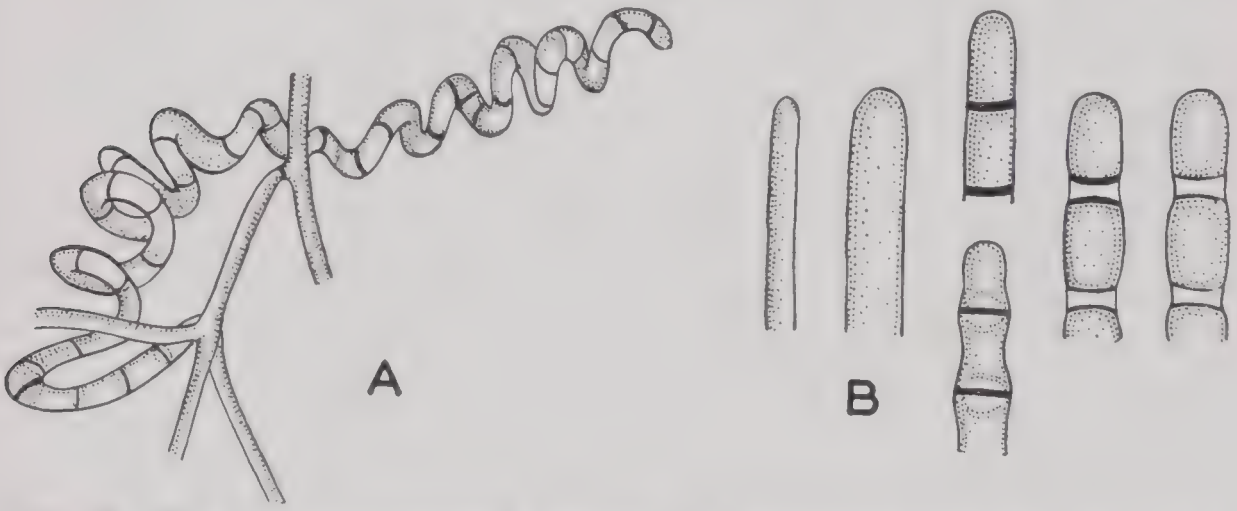

FIG. 28. The common-potato-scab organism. *A*, sporiferous coils of rudimentary hyphae; *B*, stages in the formation of spores. (*After Drechsler.*)

results in the stimulation of cambium cells to become hypertrophied and the walls to become suberized. As the fungus progresses, activity continues until a lesion is formed. The extent and type of reaction varies with the host variety, with the environal conditions, and possibly with the strain of the parasite. The larvae of soil insects are common invaders of scab lesions, and they are responsible, in part at least, for the formation of deep lesions.

Various soil factors influence the severity of the disease. One of these which has received much study is soil reaction. Numerous experiments have shown that the extent of infection is reduced quite rapidly as the pH decreases below about 5.2. There is some evidence that above about pH 8.0 infection is also inhibited. Within the range pH 5.2 to 8 the severity of the disease is little influenced, although it tends to increase somewhat up to about pH 7.0. There are numerous records of some infection at pH 5.0

or less. This has been explained by some as due to the presence of a strain of the pathogen highly tolerant to acid. However, it is rather generally accepted that when potato soils are kept at pH 5.0 to 5.2, the disease is usually under commercially satisfactory control.

Shapavalov (36) found that the spores germinated most rapidly at 35 to 40°C., while the cardinal temperatures for growth were maximum, 40.5°; optimum, 25 to 30°; minimum, 5°. Jones *et al.* (18) in a study of the relation of soil temperature to disease development found the range to be between 11° and 30.5° with an optimum at about 20 to 22°. Sanford (32) noted in 1923 that increase in soil moisture tended to reduce scab, and this was confirmed by Martin (26). Dippenaar (10) studied the relation of temperature, moisture, and soil reaction on scab development. He found the optimum temperature for infection to be slightly below 20° and that for lesion enlargement to be slightly above 20°. He also found that scab decreased with increase in soil moisture. Goss (14) emphasized the importance of these soil factors and of aeration in their effect on the soil flora for a period before infection. In some experiments the largest amount of scab occurred in soil held at high moisture content for some months before infection. Lack of soil aeration for a period before infection depressed the disease more than poor aeration provided during the postinfection period. Starr *et al.* (37) in Wyoming, in a study of the effect of different irrigation schedules on incidence of scab, found most disease in the schedule which gave the highest total water supply and the least variation of soil moisture.

The reduction of scab by certain rotation schedules has been noted by some workers. Millard and Taylor (29) in England emphasized the value of green manure as a scab depressant. They and Sanford (33) were of the opinion that the pathogen was inhibited by the activity of competitive organisms. Goss and Afanasiev (15) compared the effects of rotations on scab under irrigation in western Nebraska. They found that 4- or 6-year rotations with alfalfa were most satisfactory.

It appears at present that soil reaction and soil moisture are the most influential factors on seasonal and regional incidence of scab. But it is equally apparent that the interaction of these with other less clearly defined factors makes too rigid generalization unwise. By and large, scab remains an important disease because clear-cut, workable, reliable soil-management procedures are not yet defined.

Waksman (41) pointed out that some strains of the pathogen grew in culture media at pH values as low as 4.8. Millard and Burr (28) noted variability in pathogenicity of strains of the scab pathogen. They set up several new species and believed that some caused shallow and others deep

scab. Bruyn (5) (1939), in Holland, was inclined to the same view. Afanasiev (1) (1937), in Nebraska, was of the opinion that the production of shallow, russet, and deep scab was a function of the degree of virulence rather than of type of pathogenicity. Leach *et al.* (22) described two physiologic races on the basis of reaction to a seedling and to the resistant variety, Jubel. The seedling was very susceptible to race 1 and highly resistant to race 2, while Jubel was only moderately susceptible to race 1 and highly resistant to race 2. Schaal (35) showed that most isolates are unstable in pure culture, variants being produced frequently which differ in physiology, morphology, and pathogenicity. This was confirmed by the work of Thomas (40). Taylor and Decker (39) reported a close correlation, in 143 isolates studied, between pathogenicity and formation of a dark brown ring of surface growth on milk. It is obvious that the scab pathogen is an exceedingly variable organism; this fact may account in part for the complexity of its reaction to environmental conditions.

Varietal Susceptibility. For a long time it has been known that varieties differ in their susceptibility to scab. In some strains in which tubers have russeted skin fewer lesions appear than in smooth strains of the same variety, *e.g.*, Russet Rural, Russet Burbank, and Russet Sebago. There is reason to believe, however, that the apparent resistance is not due to a smaller number of infections but to the fact that the reaction of the cells near the surface is more limited and the lesions are thus less conspicuous. Some varieties developed in Germany and other European countries, *e.g.*, Jubel, Hindenburg, Ostragis, and Arnica, are very highly resistant. They are of no value in American potato culture as such but have been used widely as parents by potato breeders. Scab-resistant introductions derived from those parents include Menominee, Ontario, Seneca, and Cayuga.

Longrée (23) found a correlation of resistance with degree of suberization of the lenticel meristem and with a tendency of the maturing tuber to slough the lesion. Darling (9) found the lenticels of susceptible seedlings larger and made up of rounder and more loosely arranged cells than those of resistant seedlings, while the periderm of the latter suberized earlier and the suberization extended farther into the lenticel. Clark *et al.* (6, 7) found some varieties, *e.g.*, Green Mountain, to breed true for susceptibility while Hindenburg and Ostragis were apparently homozygous for resistance. The susceptible variety Katahdin carries at least one gene for resistance in a heterozygous condition. Russeting was found to be linked genetically with resistance. Stevenson *et al.* (38) tested 22 varieties in scab gardens in Maine, Michigan, Minnesota, and Colorado

during two successive seasons and found the resistance and susceptibility reactions to be fairly constant, indicating that variability in the pathogen would not seriously hamper the development of resistant varieties.

Control. Treatment of potato seed tubers for control of scab was initiated by Bolley in North Dakota in 1891. Many experiments on tuber treatment have been conducted in various parts of the United States (see Chap. 16) and elsewhere. Formaldehyde and mercuric chloride have been used extensively. The original formulae usually recommended were 1–1,000 mercuric chloride soak for 1½ to 2 hr., or formaldehyde (40 per cent solution) 1–240 soak for 2 hr. In order to shorten the period of treatment, the hot formaldehyde treatment (1–120 at 50°C., 4 min.) and the acid-mercury dip (1–1,000 mercuric chloride plus 1 per cent hydrochloric acid, 5 min.) were devised. The danger of tuber or sprout injury is greater in the shorter methods. Organic-mercury dips have been devised also, Semesan Bel (12 per cent hydroxymercurinitrophenol and 2 per cent hydroxymercurichlorophenol) being the most common. While tuber treatment is effective in reducing or eliminating introduction of inoculum, the organism has become so widely prevalent in many commercial potato soils that the benefit of tuber treatment is often of doubtful value.

Where soil is naturally rather highly acid, scab is commonly held in abeyance by use of acid-tolerant crops in rotation and avoidance of the use of lime beyond that amount which would raise the pH above 5.2. The use of sulfur on soils with higher pH has been shown to reduce scab, but except in very special cases it has not been adopted as a commercial procedure.

The effect of rotation and green manuring has been discussed in a previous section. In a few situations procedures have been worked out which are reliable, but in the main this type of remedial measure finds little application.

Resistant varieties are the ultimate solution for control of this disease. The tolerant varieties are useful in this regard, but they are not successful under conditions extremely favorable for scab. The scab-resistant introductions listed above are all late-maturing varieties, and the extent to which they may fit into potato production has not been determined (1949).

Scab remains one of the most important and least satisfactorily controlled of potato diseases.

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Powdery Scab of Potato

CHAPTER 5

DISEASES INCITED BY PLASMODIOPHORALES

The Plasmodiophorales is a small group of intracellular parasites which have a plasmodial vegetative stage; that is to say, the thallus is a naked, amoeboid, multinucleate protoplast. Each of the resting spores formed from the plasmodium within the host cells is provided with a wall. The spores germinate to release usually a single biflagellate swarm spore, which, after a period of swimming, may lose its flagella and become a myxamoeba. These small amoeboid cells may penetrate the host. In at least some forms it has been reported that swarm spores fuse in pairs to produce amoeboid zygotes before penetration of the host is effected. Many details of development are as yet obscure. The Plasmodiophorales includes some parasites on algae and phycomycetous fungi, but the majority of the species are parasites on higher plants. The order contains one family, the Plasmodiophoraceae, and eight genera. Only two species incite diseases of economic importance. Karling (3) has recently published a comprehensive monograph on the group.

The relationships of the Plasmodiophorales are uncertain, and they have been assigned various positions in schemes of classification. Many years ago Schroeter and Saccardo included them as a main subgroup under the Myxomycetes, which they considered as a class of the fungi. Fitzpatrick (1) has relegated the group to the rank of a family, which he includes in the order Chytridiales of the Phycomycetes. Sparrow, Martin, and Clements and Shear also include them among the phycomycetous fungi, while Gäumann and Dodge (2) place them in the class Archimycetes. In a recent textbook Smith (4) sets up the Myxothallophyta as one of several new divisions of the plant kingdom and includes in it the classes Acrasieae, Myxomycetae (slime molds), and Phytomyxinae, the latter class comprising the single order Plasmodiophorales.

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CLUBROOT OF CRUCIFERS

Records of this disease go back as far as the thirteenth century in western Europe. By 1800 it was widespread on turnip and rutabaga in England.

In 1872 it had become so destructive on cabbage in the vegetable gardens around St. Petersburg, Russia, that the Royal Russian Gardening Society offered a prize for a solution of its cause and control. The first intensive study on the nature of the disease was the classical work of Woronin. His first report was in 1874. A full paper appeared in Russian in 1877 and in German in 1878 (21). Clubroot is now known in many parts of the world, but it is not as yet reported from China and is quite rare in the southern and southwestern parts of the United States. Only members of the mus-

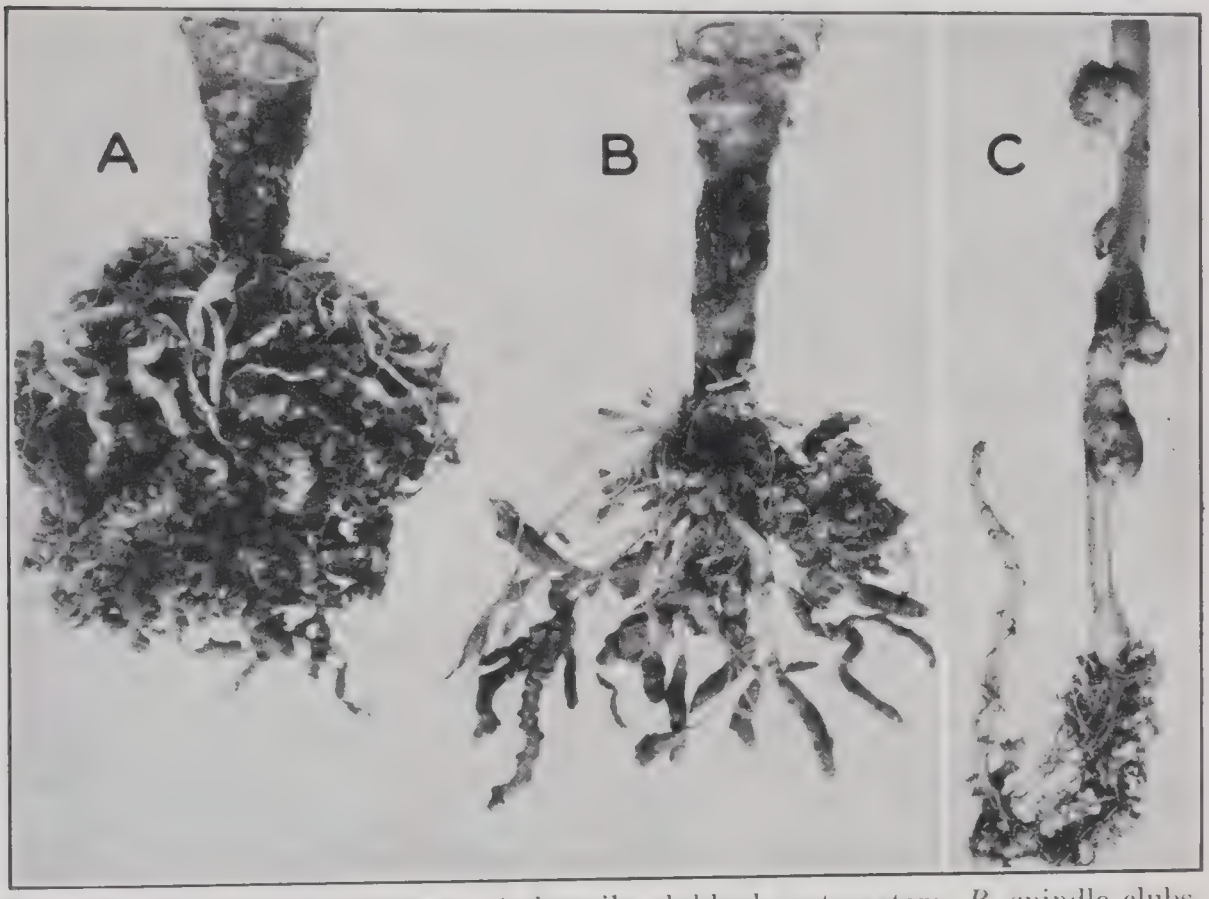

FIG. 29. Clubroot of cabbage. *A*, heavily clubbed root system; *B*, spindle clubs, most of which arose from a single infection; *C*, stem galls, which result from infection at freshly wounded leaf scars. (*After R. H. Larson.*)

tard family (Cruciferae) are affected, but within this family a large number of wild and economic species are susceptible.

Symptoms. The disease may progress to a considerable extent before above-ground signs are noticeable. Infected roots enlarge relatively rapidly to form "clubs" which take on a variety of shapes (Fig. 29). In some hosts, such as cabbage and close relatives, individual infections on roots progress in both directions parallel to the long axis, and spindle-shaped clubs result. Multiple infections bring about a variety of modifications of the spindle type. In some hosts the clubs are more spheroid than spindle-shaped. In fleshy roots, such as those of radish, turnip, and rutabaga, the

portion which is enlarged hypocotyl is not as readily infected as that which is enlarged root, where the zones of secondary roots provide favorable loci for invasion. In globe varieties, therefore, clubs are chiefly on the non-fleshy taproot or branches of it, while in long-root varieties the lower portion of the fleshy root often is affected. The rapid growth of club tissue is inimical to the normal development of cork cambium at the surface, and secondary invasion by low-grade parasites from the soil results in early decay. Soft-rot bacteria are commonly involved. The decay of clubs leads to formation of materials toxic to the plant, especially in cabbage and cauliflower, and is one of the causes of wilting of the tops. The greatest

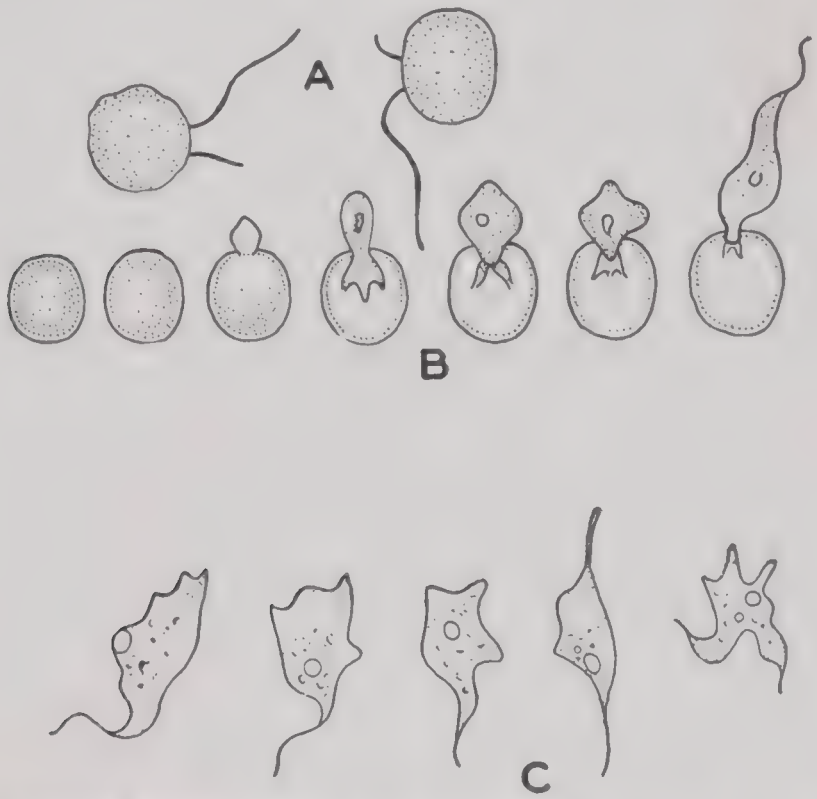

FIG. 30. *A*, biciliate swarm spores of the clubroot organism; *B*, stages in spore germination; *C*, amoeboid stage. (*A after Ledingham, B and C after Woronin.*)

source of loss to root crops is the smaller clubs, which become the avenue of invasion of rot-producing organisms responsible for decay in pits or storehouses.

The first symptoms on above-ground parts vary with environmental conditions and with the host. Sometimes temporary flagging of leaves in the middle of bright days is the first sign, particularly in cabbage and cauliflower. Such plants may wilt permanently later, or they may recover to a condition of decidedly retarded growth. Gradual and relatively inconspicuous stunting may be the only evidence of disease above ground.

The Causal Organism. *Plasmodiophora brassicae* Wor., 1877. Woronin (21) described quite fully the life cycle of the causal organism and its

relation to the host tissue. Since it resembled in many respects the saprophytic slime molds, he regarded it as a member of that class. The resting spore is hyaline, spherical, and up to 4 μ in diameter. It germinates in a favorable medium without a prolonged resting period, swelling to several times the original size and giving rise usually to a single zoospore, which

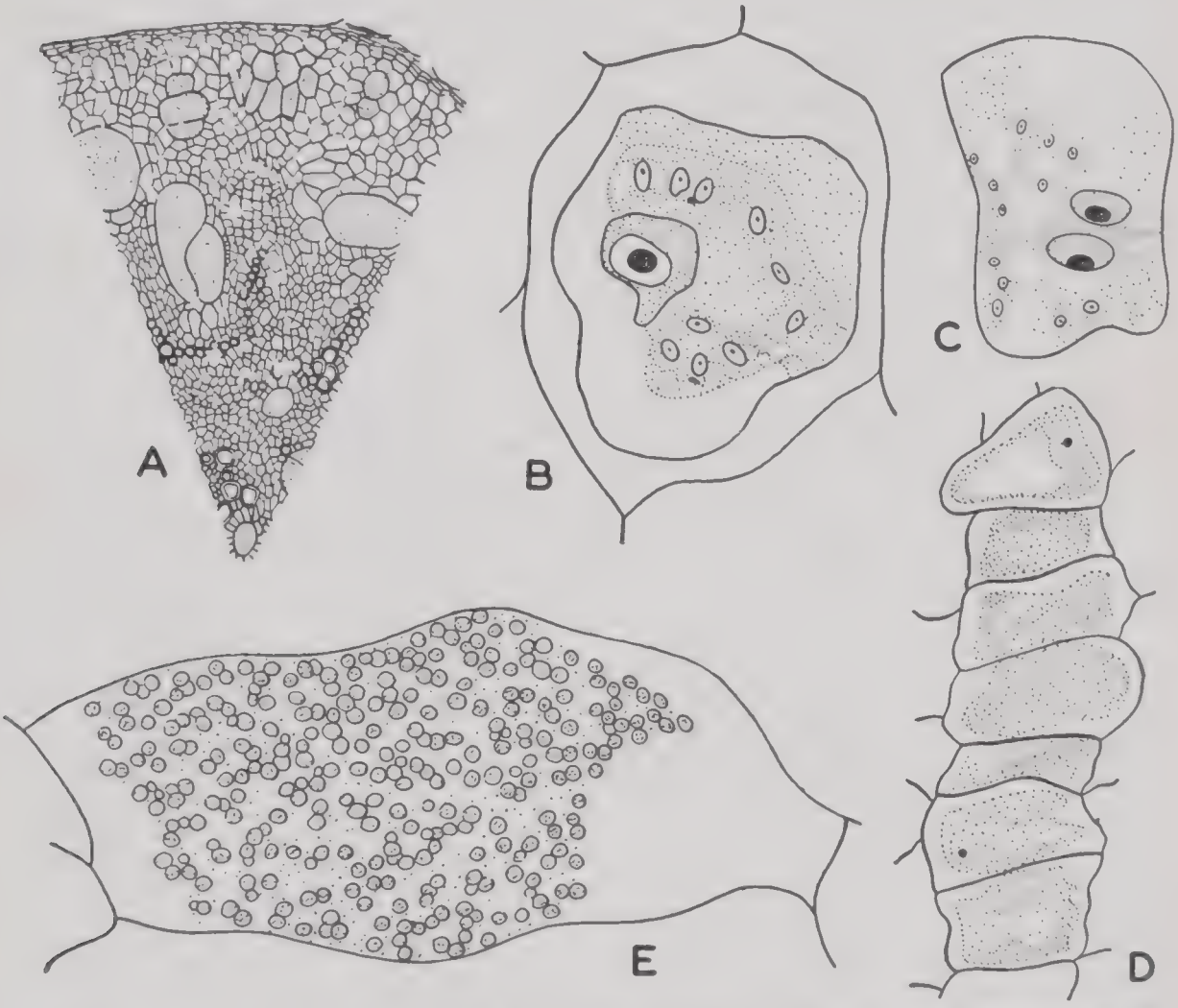

FIG. 31. Relation of the clubroot organism to the root tissue. *A*, cross section of clubbed root showing location of hypertrophied invaded cells; *B*, multinucleate plasmodium surrounding the host-cell nucleus; *C*, invaded cell in process of division; *D*, series of invaded cells arising from successive divisions as in *C*, forming a krankheitsherd; *E*, mass of mature spores in a hypertrophied cell. (*A after Woronin; B–E after Lutman.*)

emerges through a rift in the cell wall. The zoospore, a naked uninucleate protoplast, is usually exceedingly active, moving by irregular jerks for several minutes and then coming to rest or assuming an amoeboid motion. Woronin described a single flagellum having a length several times that of the spore. This has been assumed to be the case by all writers up to 1934 when Ledingham (9) demonstrated that there is a second shorter flagellum (Fig. 30).

Woronin found young amoebae in the root hairs and cortical cells of roots of cabbage. Since it is common for the zoospores of the saprophytic slime molds to fuse before the development of the plasmodium, it has been postulated by some workers that the swarm spores of the clubroot fungus fuse before penetration. This has never been proved, and convincing evidence of the penetration stage is lacking. In the host tissue the plasmodium, always within a host cell, may be found in various stages, from one with very few nuclei to a large multinucleate one which fills the entire lumen of an abnormally large cell. At no time does the plasmodium have a wall of its own, and no fruiting body is formed. In the last stage, cleavage takes place around each nucleus in the plasmodium, and separation into resting spores follows. The spores are entirely free from one another, and they are held together by the host-cell wall, until it is decomposed in the soil by secondary organisms (Fig. 31).

A number of cytologists have studied the nuclear history of this species. Since there is as yet no conclusive evidence available, the various findings and interpretations will not be presented here. An excellent review of the subject is given in the monograph by Karling (4).

Disease Cycle. Woronin (21) pointed out that the fungus was intracellular and that it had an immediate effect upon the invaded cell and a profound influence upon the nearby uninvaded cells. The invaded cell often grows rapidly to a giant size, and during this growth it may also divide to form daughter cells. In the process of host-cell division the plasmodium divides, a part going to each daughter cell. As this process is repeated, a group of invaded, enlarged cells takes form, and each eventually contains a mass of spores. To such a group of hypertrophied cells Woronin gave the name **krankheitsherd.** In addition to this host reaction an equally important one is the stimulation of parenchyma cells in the cortex and in the vascular system to divide and to continue repeated division of daughter cells. This reaction of the host, hyperplasia, is responsible in a large measure for the formation of clubs.

A much more comprehensive picture of the relation of parasite to host is provided by the work of Kunkel (5) and by that of Larson (7). While the fungus penetrates young root tissue readily, it does not invade secondarily thickened roots or subterranean hypocotyl unless wounds are provided (7). It will invade the lower stem, above ground, through needle wounds or through fresh leaf scars (7). When the fungus has invaded the roots, it migrates from cell to cell as amoebae or small plasmodia, and in these stages probably stimulates most of the hyperplasia. In those cells in which the plasmodia stimulate giant-cell formation and krankheitsherde, the fungus probably is relatively immobile. As the migrating form moves, it produces hyperplasia in the cortex or periderm, but, in the root, the most

reactive tissue is in the cambium. Here excessive proliferation of cells is stimulated, and also normal differentiation of cells into xylem and phloem is retarded and largely prevented. After the fungus reaches the cambium, it moves upward and downward in this tissue more rapidly than it does in the cortex, but hyperplastic activity at the point of invasion of the cortex and of the cambium continues. There follows, naturally, a tendency for the diseased root to assume the shape of a spindle in which the greatest diameter is at the point of invasion, and size tapers off in both directions parallel with the long axis of the root (5). Spindle-shaped clubs, common in cabbage, are the result of single-point infection. When there are multiple infections in close proximity, the spindle-shaped clubs are less conspicuous. In the stem, the tendency for spindle formation decreases as the point of infection passes above the cotyledonary node. When infection is at fresh leaf scars, the clubs tend to be spheroid galls rather than spindles (Fig. 32). Cunningham (2) has shown that the gross anatomy of clubs varies with the host species.

Infected subterranean plant parts disintegrate in the soil, releasing the free spores. While the spores germinate with little or no resting period, there is good reason to believe that a large percentage of them remain dormant for many years. There is no evidence that the fungus lives as a saprophyte in the soil or elsewhere, yet soils are known to remain infested for 10 years or longer without the presence of host plants. The fungus is one of the most persistent soil invaders, and crop rotation is of little value in control. It was observed before the middle of the nineteenth century that application to the soil of alkali-producing materials, such as wood ashes or lime, reduced infection. Liming of soils became a control practice in many areas, but in some cases it was not consistently effective. It has been shown that the spores germinate poorly or not at all in an alkaline medium. Wellman (20) and Larson and Walker (8) showed that on silty clay soils in southeastern Wisconsin the disease was often destructive after the soil had been limed to the point where, by usual methods of analysis, the pH of the soil solution was above pH 7.0. When they brought such soils to the greenhouse, however, and grew plants in them with daily watering, no disease developed (8). They interpreted this by postulating that in the field the rhizosphere zone immediately adjacent to fibrous roots might have a lower pH than the soil mass, owing to excretion of carbon dioxide by the roots, and that the spores germinated and penetrated at these loci uninhibited by hydroxyl ions. In frequently watered soils there would be less opportunity for the maintenance of pH levels in the rhizosphere favorable to germination and infection. On muck soils with high buffer capacity, liming is of little value in reducing infection.

Monteith (10) found infection to occur at soil temperatures from 9 to

30°C. and did not believe soil temperature ordinarily limited infection. He found that infection was limited by low soil moisture, and it was prevented in plants grown in the infested soil he used if the water content was not allowed to rise above 45 per cent of the water-holding capacity. This was in accord with a common observation that the disease is often most severe in, and sometimes confined to, low poorly drained portions of fields. Wellman (20) later showed, however, that if the moisture content of dry infested

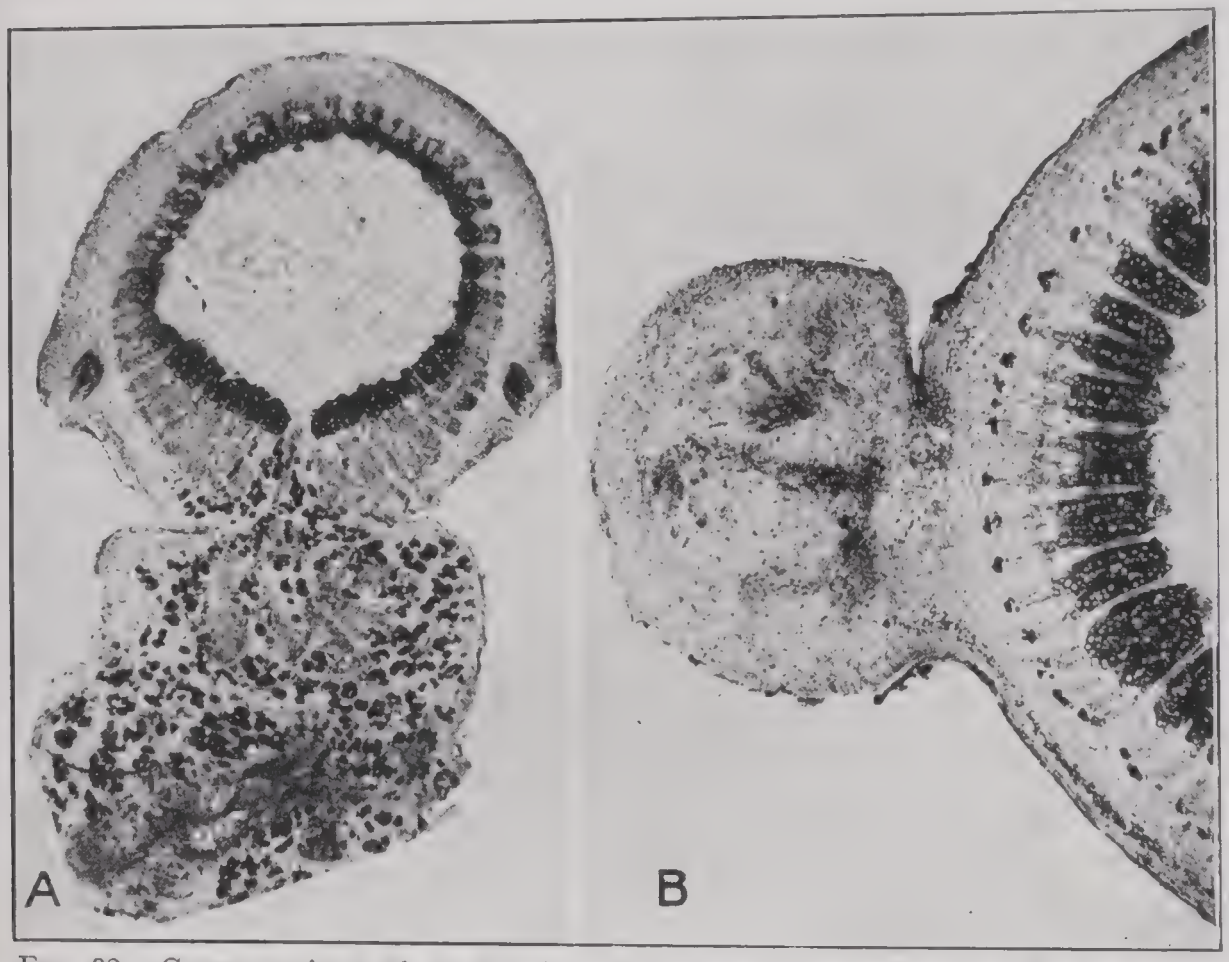

FIG. 32. Cross sections of stem galls. In *A* some hyperplasia in the cambium occurred, but most of the gall is derived from cortical tissue. The dark areas are krankheitsherde containing masses of mature spores. In *B* the gall is entirely cortical in origin. (*After R. H. Larson.*)

soil was raised to the optimum moisture for 18 to 24 hr., germination and penetration followed, and disease development was not affected by soil moisture after infection had occurred. This helped to explain the occasional observation that more disease occurred in high than in low areas in a given field. Pryor (11) and Walker and Hooker (18) working with sand nutrient cultures showed that the disease was reduced by low potassium and low phosphorus. Low nitrogen, high nitrogen, high potassium, and increase in concentration of a balanced nutrient enhanced disease development.

The organism is disseminated locally with drainage water, farm implements, wind-borne soil, roving animals, and transplants. It does not gain access to the seed and is, therefore, not seed-borne. Transplants comprise the chief means of widespread distribution.

Host Resistance. The most extensive work on development of resistant varieties has been that with turnip and rutabaga in Denmark, Sweden, and Great Britain. Resistant varieties of stock turnip in commercial use include Bruce, May, and Dale's Hybrid. Resistant varieties of rutabaga are Wilhelmsburger, Resistant Bangholm, and Immuna II (6). In England, certain varieties of marrow kale have been found to be sufficiently resistant to be of commercial value. Walker (15) found that most varieties of turnip and rutabaga were resistant to the fungus as collected from various regions in the United States, although some of the same varieties were reputed to be less resistant in Europe and in eastern Canada. Although several workers had indicated the probability of distinct physiologic races of the fungus, this was not proved until Walker (16) showed that in certain varieties of turnip completely resistant in inoculation tests in Wisconsin, a considerable percentage of plants were infected when portions of the same seed lot were grown in England. No resistant varieties of cabbage or cauliflower have been developed, and all strains and varieties tested have been completely susceptible to the United States strain of pathogen. Recent hybridization experiments with a naturally resistant kale-cabbage hybrid offer the possibility of developing commercially acceptable resistant varieties of cabbage (19).

Black mustard is commonly listed as a resistant host. Walker (14) showed that when collections from various parts of the world were tested, the lots varied from highly resistant to very susceptible. It was claimed by Rochlin in Russia that resistance in black mustard was due to the volatile mustard oil in that plant. This contention was disproved by Stahmann *et al.* (13) and Hooker *et al.* (3). Pryor (11) showed further that when resistant mustard was grown in a sulfur-free nutrient, the volatile sulfur oil did not occur but resistance was not altered.

Control. Since transplants are the most common means of local and widespread distribution of the fungus, it is important to use soil free from the pathogen in propagation of seedlings. In many areas liming of the soil is used to minimize damage, although this practice has limitations which have been discussed under Disease Cycle. Where potatoes are grown in rotation with crucifers, liming is inadvisable, since it provides conditions which are particularly favorable for common scab. In general, gardeners choose to avoid land for susceptible crucifer crops after it has become infested with the clubroot organism. Resistant varieties of stock turnip and of rutabaga are available, but much more improvement in resistance in

these and other cruciferous crops is needed. The use of mercuric chloride (1-1,500) at the rate of 125 ml. per plant as the transplanting liquid for cabbage and cauliflower has some protective value (17).

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POWDERY SCAB OF POTATO

This disease is of interest because it is the second most important one incited by a member of the Plasmodiophorales, and because of its origin and distribution. It was first found in Germany in 1841, where it was described by Wallroth the following year. By 1885 it was widely distributed in Europe. In 1891 it was reported to be rather generally distributed in the vicinity of Quito, Ecuador, and has since been recorded in Colombia, Peru, and Chile. This is probably its original habitat whence it was transported to Europe with the potato. It has become established in Russia, Armenia, Morocco, Algeria, Kenya, Madagascar, South Africa, Rhodesia, New Zealand, Australia, and Hawaii. The first report of the disease on the North American continent was by Güssow (2) in 1913, who reported having received specimens from eastern Canada. In the same year it was found in northern Maine in an area contiguous to the Canadian province of New Brunswick. An extensive survey in the United States from 1913 to 1915 showed it to occur in a very minor degree in widely scattered areas in New York, Florida, Minnesota, Oregon, and Washington. There is good evidence that it had been present in Maine for 15 years or more before it was discovered.

The fungus was shown, experimentally, by Melhus *et al.* (8) to incite galls upon the subterranean parts of tomato as well as those of several species of *Solanum*. It was later found to infect *S. nigrum* L. (3).

Symptoms. The first symptom on potato tubers consists in faintly brown raised areas, about the size of a pinhead, which enlarge in 6 to 8 days to about $\frac{1}{2}$ cm. In the meantime the brownish color disappears, and the diseased tissue becomes jelly-like. It soon contains the mature yellow-brown spore balls of the fungus. The epidermis rifts and peels back to expose the dusty spore mass formed in the sorus (Fig. 33). On roots of potato, tomato, and other hosts the proliferated tissue causes distinct galls, which vary in size up to that of a pea. The sori on tubers sometimes enlarge to form cankers. In storage, dry rot ensues from the sori, owing in part to a semisaprophytic growth of the fungus and in part to invasion by secondary facultative parasites.

The Causal Organism. *Spongospora subterranea* (Wallr.) Lagerheim, 1892. The fungus was described in 1842 by Wallroth as *Erysibe subterranea*. No further critical study was made until that by the Norwegian botanist, Brunchorst, who noted plasmodia in potato cells and placed it in the Plasmodiophorales as *Spongospora solani*. Lagerheim (5) restored the original species name, *S. subterranea*. Johnson in Ireland described germination of the spores. Kunkel (4) worked on the life history of the fungus. The skin of the potato is penetrated by a plasmodium, which kills some cells at the point of entry and passes through and between the epidermal

cells, spreading out in a disk-shaped mass. Wild (11) considered lenticels and wounds to be the chief avenue of invasion. Cell division is stimulated in advance of cell invasion, and a new periderm is formed, which in turn may be invaded. The fungus moves as the plasmodium between the host cells and penetrates the softened walls to invade the cells. Some invaded cells grow to several times the normal size, and most of the growth is near the periphery of the tuber, contributing to the characteristic raising and eventual rifting of the epidermis. The giant cells later divide into several infected cells about the size of normal cells. On roots, the galls are produced by the same type of cell enlargement and division, particularly in the phloem and meristematic tissue. The plasmodium is multinucleate. Ledingham (6) describes zoosporangia, which occur singly or in clusters up to a dozen or more within the host cell; they are variously shaped, hyaline, and thin-walled, giving rise to zoospores which penetrate the host-cell wall. Resting spores are formed from the plasmodium in spore balls or clusters (19 to 85 μ), which are spongy with irregular internal channels. They form a yellow-brown dust in the mature sori. The spore ball contains many individual cells, each constituting an individual uninucleate spore. Each spore upon germination sets free a swarm spore which is indistinguishable from that produced in the zoosporangium. They are spherical, 2.5 to 3.5 μ, with two unequal flagella.

FIG. 33. Powdery scab of potato. (*Courtesy of Donald Folsom.*)

When dry rot sets in around mature sori, newly formed plasmodia penetrate the dormant tissue. There is little or no host reaction in the form of cell divison and growth, and as the cells are penetrated and killed, there develops a disorganized mass of dead cells which constitutes the dry rot. Kunkel (4) regards this phase as a purely saprophytic development of the fungus. He grew plasmodia saprophytically on agar media but did not produce infection with such cultures.

The entire life cycle of the fungus is still not clear. Whether or not the swarm spores grow saprophytically in the soil, whether they fuse to produce plasmodia, and whether swarm spores from resting spores and from zoosporangia are distinct or similar in their nuclear make-up remain to be determined. Some workers state that there is a meiotic division of nuclei, just before cleavage of the plasmodium to form resting spores.

Disease Cycle. The organism persists for undetermined periods in soil under certain conditions. The world history of the disease indicates that the pathogen becomes endemic, however, within a limited range of environal conditions. Ramsey (10) pointed out that in 1916, when weather was unusually warm and dry in Maine, the disease did not appear in fields where it had occurred previously. In the greenhouse, it developed on tubers grown in infested soil only when the temperature was not above about 14°C. and when the soil moisture was relatively high. When the disease appeared in Maine, there was naturally much concern as to whether large annual shipments of seed potatoes from that state would result in a prompt dispersal and establishment of the pathogen in many other important potato-producing regions. Experimental tests by Melhus *et al.* (8), in which infected tubers were planted in several Atlantic seaboard states from Massachusetts to Florida, resulted in no infection in the resulting crops. When the soils from the plots in which the plants grew were transported to a virgin field in Maine and when potatoes were grown upon them, infected tubers resulted in most of them. This implied that the fungus was introduced and established but was not infectious in the areas selected. It was primarily on the basis of these results that the temporary quarantine of Maine potatoes was lifted. The fact that the disease has remained a rare and unimportant one in the United States is good evidence that the fungus is restricted by a narrow range of optimum environment. This is in direct contrast with the ring rot of potato, which appeared some years later in eastern Canada and in Maine and very promptly became a serious menace to potato production throughout the United States.

Control. The importance of the disease depends upon a favorable environment and infested soil. No control measures have been worked out that are generally satisfactory. In Germany, Russia, and Chile attention has been given to screening varieties to select those which are highly resistant. Since the disease is not important in the United States, no emphasis has been given to development of resistant varieties. Standard tuber treatments (p. 152) reduce the amount of inoculum carried on seed tubers, but owing to the fact that spores and plasmodia are deep seated in tuber tissue, the treatments are not completely effective.

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CHAPTER 6

DISEASES INCITED BY PHYCOMYCETES

The Phycomycetes are a rather heterogeneous group of some 1,500 species and are commonly referred to as the lower true fungi, since they seem to stand at a lower evolutionary level than the Ascomycetes, Basidiomycetes, or Deuteromycetes. Many species in the group are parasites upon algae, other fungi, or upon seed plants; among these are the incitants of some important plant diseases. Other species appear to be strict saprophytes. The more advanced members of the Phycomycetes have a well-developed **coenocytic** mycelium (*i.e.*, the plant body consists of a system of hyphal threads which are multinucleate and typically nonseptate). In lower forms, most of which are aquatic, the thallus is very simple and often does not have a mycelial organization. This is the case in most of the Chytridiales, one of the lowest orders in the group. Furthermore, the simplest members of the Phycomycetes are **holocarpic,** which is to say that the protoplasmic content of the entire plant body is used up in reproduction. In the more advanced members, on the other hand, special reproductive organs are differentiated on the mycelium.

A majority of the members of the class produce motile spores (**zoospores,** or **swarm spores**) in the process of vegetative reproduction. In forms with a mycelium these are borne in **zoosporangia** differentiated most typically at the ends of hyphae. In holocarpic types the entire thallus may function as a single zoosporangium, or the protoplast may divide up to form a number of zoosporangia. In the order Mucorales most species produce sac-like sporangia, within which are formed nonmotile **sporangiospores.** The hypha or system of hyphae which bears sporangia—whether these form nonmotile spores or zoospores—is commonly known as a **sporangiophore,** if it is a clearly differentiated unit distinct from the vegetative hyphae system.

In the Peronosporales the zoosporangia are for the most part deciduous. Such deciduous sporangia are commonly referred to as **conidia** (singular, **conidium**) and, correspondingly, the hyphae which bear them are termed **conidiophores.** In most cases these detachable sporangia may germinate either **indirectly,** *i.e.*, producing zoospores, or **directly,** *i.e.*, with the formation of a germ tube, depending apparently largely upon the environal conditions. However, in *Peronospora*, which is often regarded as the most advanced genus in the group, only direct germination is known to occur. Throughout the Entomophthorales conidia occur, and conidia are produced also by a few members of the Mucorales.

Sexual reproduction in the Phycomycetes is as varied as is asexual reproduction. Among holocarpic forms the protoplasts of two entire thalli may fuse to form a resting spore, or small motile gametes may be formed, these uniting in pairs in the water to form motile zygotes. The latter situation undoubtedly represents the most primitive form of sexuality in the entire class. In the water molds and in the Peronosporales small male sex organs termed **antheridia** (singular, **antheridium**) and larger female sex organs termed **oogonia** (singular, **oogonium**) are formed on the mycelium. One or more eggs are differentiated within an oogonium. A **fertilization tube** then grows into the oogonium from the antheridium, and one or more male nuclei are discharged into each egg. Nuclear fusion occurs, and from each fertilized egg a thick-walled oospore is formed. In some forms, apparently, the eggs may develop into thick-walled resting spores without fertilization. It has been suggested that the name **aboospore** be applied to these parthenogenetically developed structures. In the Mucorales two multinucleate **gametangia,** which are often almost identical in size and shape, fuse to form a thick-walled **zygospore.** In this case, then, no gametes are differentiated, and the fusing gametangia are not clearly sexually different. However, in many mucors it has been shown that for zygospore formation two different mycelia must be brought together and that one of the gametangia of a fusing pair is formed on one mycelium while the second gametangium is produced on the other mycelium. The phenomenon involved here is known as **heterothallism,** and species which exhibit it are said to be **heterothallic.** The two kinds of mycelia are referred to as "plus" (+) and "minus" (−). A (+) mycelium commonly is slightly more vigorous than a (−) mycelium of that same species, but there is ordinarily no other obvious difference. Heterothallism has been reported, too, in some of the water-mold fungi, and it is probable that further research will reveal its occurrence in phycomycetous groups, where it is not now known.

Classification of the fungi is currently in a state of flux, as is evidenced by the fact that no two textbooks on mycology follow the same scheme and by the fact that we are being bombarded by numerous proposals of new groupings. The Phycomycetes are no exception to the situation. Our knowledge, especially concerning structure and development in lower members of the group, has advanced rapidly in recent years; and new taxonomic alignments have been drawn accordingly, many of them cutting across the old subclass boundaries and in some cases even across the boundaries of old orders and families. The treatments of Sparrow (4) and Karling (3) are very modern for the portions of the class with which they deal but unfortunately are rather limited in their scope. Fitz-

patrick's book (1) on the lower fungi, although not completely up to date, is the only relatively recent and readily available comprehensive treatment of the phycomycetous fungi.

Many types and degrees of parasitism are found within the Phycomycetes. Some species are obligate parasites on flowering plants (*e.g.*, species of *Synchytrium*, *Albugo*, *Peronospora*, *Plasmopara*); others are relatively low grade pathogens commonly attacking weakened plants or dormant fleshy organs (*e.g.*, *Pythium*). Many of the chytrids and members of the Lagenidiales are parasites on algae or fungi, and certain members of the group attack various forms of aquatic animal life. Examples of the Phycomycetes treated in this chapter are selected from the Chytridiales, Saprolegniales, Peronosporales, and Mucorales. A brief key to the causal organisms treated follows.

Chytridiales. Mycelium absent or poorly developed; the thallus at maturity taken up largely in reproduction.

Synchytriaceae. Thallus either developing a thick wall and functioning as a resting spore or dividing by cleavage to form a sorus of sporangia.

Synchytrium endobioticum (Schilb.) Perc., incitant of black wart of potato.

Cladochytriaceae. Mycelium wide-spreading, developing terminal and intercalary enlargements which are transformed into sporangia or resting spores.

Physoderma zeae-maydis Shaw, incitant of brown spot of maize.

Urophlyctis alfalfae (Lagh.) Magn., incitant of crown wart of alfalfa.

Saprolegniales. Mycelium well developed and functioning in only small part in reproduction; usually aquatic; motile spores formed endogenously in sporangia; one to many oospheres in the oogonium.

Saprolegniaceae. Thallus branching without definite constrictions.

Aphanomyces euteiches Drechs., incitant of root rot of pea.

Peronosporales. Mostly terrestrial and parasitic; oogonium forms a single oosphere.

Pythiaceae. Sporangiophores not sharply differentiated from hyphae. Sporangia borne successively.

Pythium spp., *Phytophthora* spp., incitants of damping-off, root rot, and soft rot.

Phytophthora infestans (Mont.) DBy., incitant of late blight of potato and tomato.

Albuginaceae. Sporangia borne in a chain.

Albugo candida (Lév.) Kunze, incitant of white rust of crucifers.

Peronosporaceae. Sporangiophore sharply differentiated from mycelium and bearing sporangia simultaneously at tips of branches.

Sclerospora graminicola (Sacc.) Schroeder, incitant of downy mildew of grasses.

Plasmopora viticola (Berk. & Curt.) Berl. & deT., incitant of downy mildew of grape.

Pseudoperonospora cubensis (Berk. & Curt.) Rostow., incitant of downy mildew of cucurbits

Bremia lactucae Regel, incitant of downy mildew of lettuce.

Peronospora destructor (Berk.) Casp., incitant of downy mildew of onion.

Mucorales. Gametangia usually similar in size and shape, fusing to form the zygospore. Asexual spores vary; in some forms they are produced in a many-spore sporangium.

Mucoraceae. Sporangium containing a columella, wall thin and not cutinized.

Rhizopus nigricans Ehr., incitant of soft rot of sweet potato and other vegetables.

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BLACK WART OF POTATO

Black wart is an important disease of potato in Europe. It was discovered first in Hungary, where it was described in 1895. It is now known to occur in most countries of western Europe. In 1940 it was claimed not to have been found in U.S.S.R., except in territories recently acquired from Finland and Poland. It has been reported in South Africa. In the Americas it was found in the Andean region of Peru, which is the only South American country from which it had been reported up to 1944. It was found in Newfoundland and in some neighboring islands in 1909. In Canada it was reported in 1918 but apparently had not recurred until 1941, when it was found in a small garden near Halifax, Nova Scotia, in which case appropriate quarantine measures were taken. The disease was discovered in the United States in 1918 in home gardens of mining villages in Pennsylvania and later in a few similar locations in West Virginia and Maryland. Fortunately these were not commercial potato-producing areas, and quarantine measures and required planting of immune varieties have been applied since 1918. No other occurrence has been reported in this country.

Host plants besides the potato and tomato include black nightshade (*Solanum nigrum* L.), bittersweet (*S. dulcamara* L.), and several other species of *Solanum;* henbane (*Hyoscyamus niger* L.); and apple of Peru (*Nicandra physaloides* Gaertn.).

Symptoms. The disease appears on all underground parts other than the roots. Buds on stems, stolons, and tubers are the centers of infection

and abnormal growth activity leading to wart formation. The warts vary from very small protuberances to large intricately branched systems. A typical wart is roughly spherical, but it is usually not a solid structure (Fig. 34). It is soft and pulpy and can be cross-sectioned more readily than a tuber. Morphologically it consists of distorted, proliferated branches and leaves grown together into a mass of hyperplastic tissue. The underground wart is usually similar in color to a normal stolon or tuber. Often a developing wart is exposed at or above the soil line, when

FIG. 34. Black wart of potato. (*Courtesy of R. E. Hartman.*)

chlorophyll forms and the color changes accordingly. As the warts become older, when above or below ground, they darken somewhat, and decay by secondary organisms sets in. When affected tubers are dug, warts may have disintegrated in part.

The Causal Organism. *Synchytrium endobioticum* (Schilb.) Perc., 1910.

SYNONYMY: *Chrysophlyctis endobiotica* Schilb., 1895.

The organism was first described by Schilbersky, who created a new genus, *Chrysophlyctis*, to accommodate it. Percival later transferred the

species to *Synchytrium*, and the former genus is no longer valid. The most complete study of the life history (Fig. 35) is by Curtis (6). The **resting spore** is found in the wart tissue, and it also remains viable for a long period in the soil. It contains an outer brittle membrane, consisting of disorganized remains of host-cell contents, and two inner membranes. The innermost membrane is thin and hyaline. Before germination uniflagellate zoospores are formed within the innermost membrane; the membranes rupture and free the spores. The zoospore comes to rest on the host surface and germinates by a very thin naked penetration tube, which pierces the cuticle and epidermal wall of the bud tissue in stolons or tubers. It gives rise promptly to a uninucleate intracellular thallus, which grows to considerable size as the host cell becomes hypertrophied. As the thallus matures, it becomes a **prosorus,** which has a thick orange exospore and a thin hyaline **endospore.** The endospore is extruded with the cytoplasm and nucleus to form a **vesicle,** where the nucleus divides until about 32 nuclei are formed. The contents of the vesicle are traversed by newly formed walls, which delimit four to nine **sporangia;** these compose the **sorus.** Nuclei divide in each sporangium to the number of 200 to 300. The vesicle membrane and the host-cell wall are finally ruptured by internal pressure, and the sporangia are set free. Numerous uninucleate uniflagellate zoospores are set free upon the rupture of a hyaline papilla in the sporangial wall. Some of these swarm spores reinfect the host; others fuse in pairs to form a uninucleate zygote. The latter, when in contact with the host, penetrates the epidermal wall much as the zoospore does and grows within a hypertrophying host cell to form the resting spore. Curtis (6) believes that fusion occurs only between zoospores from distinct sporangia.

Disease Cycle. The resting spores of the organism remain viable in soil for many years, although longevity is influenced by the type of soil and the crop sequence. Viable resting spores were found in air-dry soil after 15 months and in dry wart material after 5 years. Since areas containing infested fields are kept under quarantine in the United States, the methods of dissemination of spores are important. Potato tubers are, of course, the most ready means. Some commercially resistant varieties are particularly liable to spread the organism because inconspicuous warts may occur which have little or no economic importance but nevertheless bear resting spores. Tubers of highly resistant varieties are not free from suspicion. Contaminated manure and infested soil transported in various ways are means of spread.

Short periods of saturation of the soil are essential for germination of resting spores and sporangia, as well as for liberation and movement of zoospores. Spores and sporangia will germinate over a wide range of

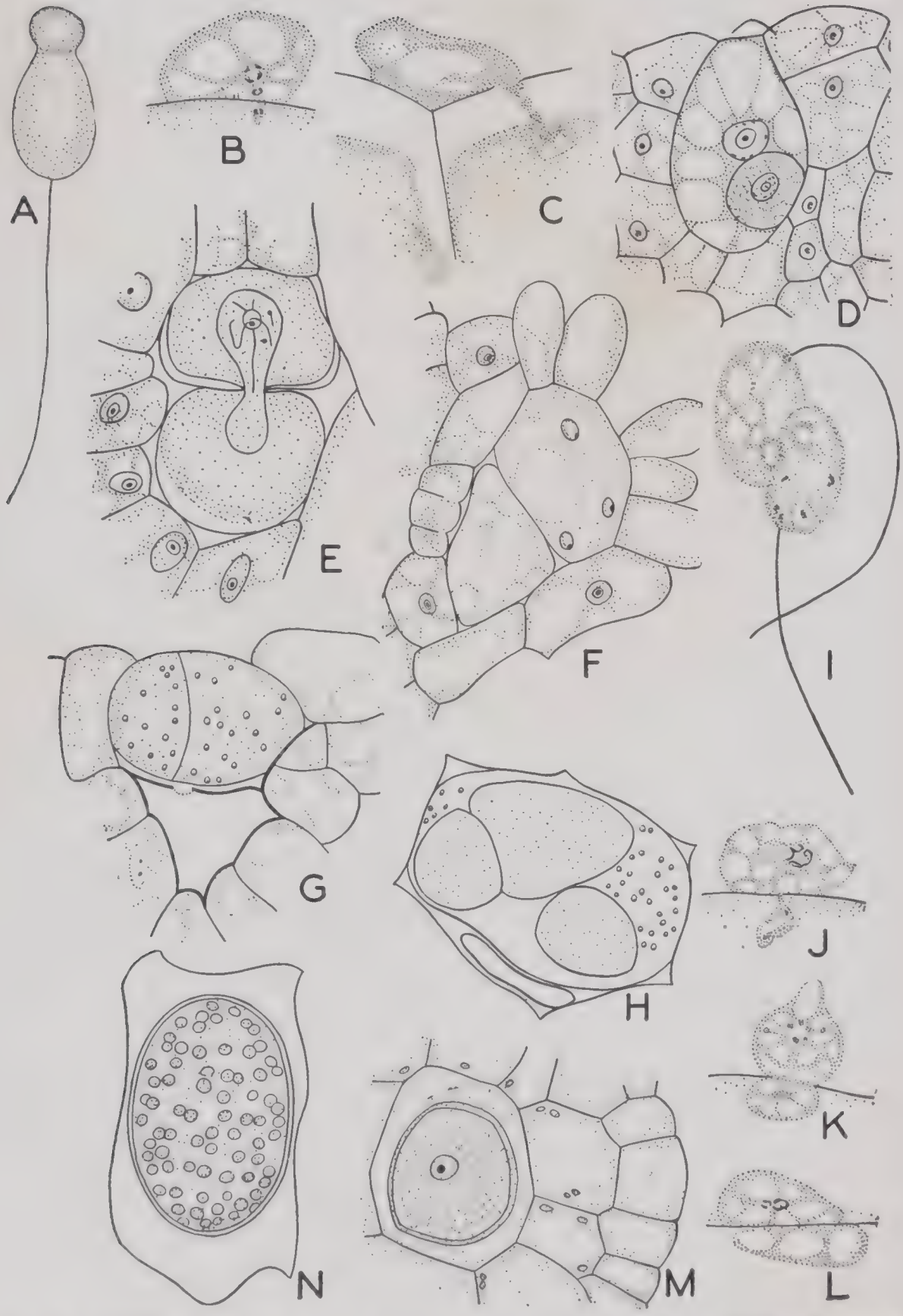

FIG. 35. Stages in the life cycle of *Synchytrium endobioticum*. *A*, zoospore from a resting sporangium; *B*, *C*, zoospore entering the host cell by direct penetration; *D*,

temperature if moisture is favorable. In experimental trials with constant soil temperature, infection was limited to a range of 12 to 24°C. Infection occurred over a range of soil reaction from pH 3.9 to 8.5, but a neutral to slightly acid soil was the most favorable (17). Movement of the organism within the soil is negligible as a factor of dissemination.

A newly infected bud exhibits numerous small pustules, which consist of protuberances in which brownish depressed centers appear. These centers are sori containing summer sporangia around which hyperplasia of the host tissue has been initiated. As the zoospores of the sporangia are discharged, reinfection occurs and new centers are formed. The wart develops as dormant buds are stimulated, and the hyperplastic mass of stem and leaf tissue forms. The anatomy and histology of the wart tissue is described in detail by Artschwager (1).

The fungus, so far as is known, is an obligate parasite.

Varietal Resistance. It was observed early in this century by potato growers in England (10) that many varieties were resistant to wart while others were extremely susceptible. Much study has been given to this subject in many of the European countries. In England, Scandinavia, and Germany extensive programs have long been under way in which standard varieties were screened for resistance and new varieties brought out by breeders were tested. While earlier methods involved growing varieties on naturally infested soil, this procedure has been greatly simplified by several laboratory tests. One commonly used consists in placing tubers which have broken dormancy in moist sand or moss and ringing an eye with petroleum jelly. Within the ring is introduced a suspension of zoospores prepared by soaking warty tissue in water. As the young growing bud is infected, wart development proceeds promptly if the variety is susceptible. Invasion of all varieties occurs, but in the highly resistant ones the pathogen dies without producing resting spores or prosori (12). It is now recognized that a wide range of resistance occurs, some varieties responding, characteristically, by production of no warts or very small ones, while at the other extreme warty proliferation is very extensive. It is the moderately resistant varieties in which warts are

young prosorus in an enlarged host cell; *E*, the contents of the prosorus passing into the host cell; *F*, a later stage than *E* with a slight amount of protoplasm still in the prosorus; *G*, a cross section of the sorus showing two sporangia with the remains of the empty prosorus below; *H*, three mature sporangia in the sorus with zoospores beginning to be liberated; *I*, two zoospores from a sporangium in juxtaposition preparatory to fusion to form a zygote; *J*, *K*, *L*, stages in the direct penetration of an epidermal host cell by a zygote; *M*, the resting sporangium in the third layer of host cells after two successive divisions of the invaded epidermal cell; *N*, resting sporangium in which maturation of zoospores, to be discharged later (see *A*), is going on. (*After Curtis.*)

readily overlooked that are most effective in spreading the pathogen, since in the small warts, which cause little or no commercial loss, resting spores are commonly produced and thereby distributed.

Black (2) has suggested that resistance is controlled by the cumulative interaction of three genes. Lunden and Jørstad (14) presented data which they interpreted to show two independent dominant genes and two other complementary genes which conferred resistance when both were present.

Blattný (3) announced in 1942 the differentiation of three physiologic races of the fungus in central Europe.

The standard varieties in common use in the United States may be divided into resistant and susceptible groups as follows:

Resistant		Susceptible	
Green Mountain	Katahdin	Rural New Yorker	Sebago
Burbank	Mohawk	Russet Rural	Golden
Russet Burbank	Sequoia	Early Ohio	Erie
Irish Cobbler	Mesaba	Triumph	Earlaine
McCormick	Norkota	Warba	Potomac
Spaulding Rose	Houma	Chippewa	

It is to be noted that some of the resistant and susceptible varieties are closely related genetically. Katahdin (resistant) and Chippewa (susceptible), for instance, were derived from the same cross.

Control. In many countries various degrees of quarantine have been practiced to reduce spread of the pathogen. In the United States the potato embargo implemented under the Federal Plant Quarantine Act has prohibited entry of potatoes from any country where wart is known to occur. The localities in the United States which contain infected soil are required to use only resistant varieties, and export of potatoes from these localities is prohibited. Distribution of the pathogen has thus been effectively prevented.

After wart was found in the United States, many experiments were conducted on soil treatment to eradicate the disease. This procedure, while possible by several means, is too costly and impracticable to apply.

Should wart appear in other areas, the only feasible control will be the use of resistant varieties. In Europe this remedial measure has been applied extensively where infected areas are more numerous and the disease correspondingly more important.

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BROWN SPOT OF MAIZE

This disease was found in India in 1910 and in Illinois in 1911. It occurs throughout the southeastern quarter of the United States. It is of some importance in upper Florida and in regions immediately to the north, but it has never become destructive in the states of the corn belt. All types of maize, *Zea mays* L., *i.e.*, field corn, sweet corn, popcorn, etc., are susceptible. Teosinte (*Euchlaena mexicana* Schrad.) is susceptible, but various other close relatives of maize which have been tested have not

been found to become infected. A related disease on quack grass, *Agropyron repens* (L.) Beauv., was reported from Wisconsin in 1947 (3).

Symptoms. The disease occurs on blades, sheaths, and culms, and only rarely on husks of the ears. The lower portion of the plant is usually the more severely affected. The first sign is the appearance of slightly bleached or yellowish spots, usually in great numbers, which turn brown promptly and seldom exceed 1 mm. in diameter on the blade and 5 mm. on the midrib or sheath. The affected plant parts assume a rusty appearance. The sheath may become reddened, and severely affected blades

FIG. 36. Brown spot of corn. (*After Dickson.*)

may die prematurely. Spots on the culms are concentrated, usually near the nodes, and they weaken the tissue sufficiently to cause lodging (Fig. 36).

The Causal Organism. *Physoderma zeae-maydis* Shaw, 1910. The brown spots on diseased plants contain smooth, brown, thick-walled spores (18 to 24 by 20 to 30 μ), which are flattened at one side where the outline of a definite lid or cap is visible. This thick-walled structure is sometimes termed a resting sporangium; often it is called simply a resting spore. On germination the lid opens, and a thin-walled vesicle extends through the opening (Fig. 37). The vesicle ruptures at the apex and liberates uniflagellate zoospores (3 to 4 by 5 to 7 μ). When these zoospores are in contact with a corn leaf in moisture, they move about for a

time and then come to rest. They germinate and infect the host cell or give rise to irregular slipper-shaped structures, varying in size, which are anchored to the host tissue by a coarse, branched rhizoidal mycelium. These structures are thin-walled sporangia, and each discharges at ma-

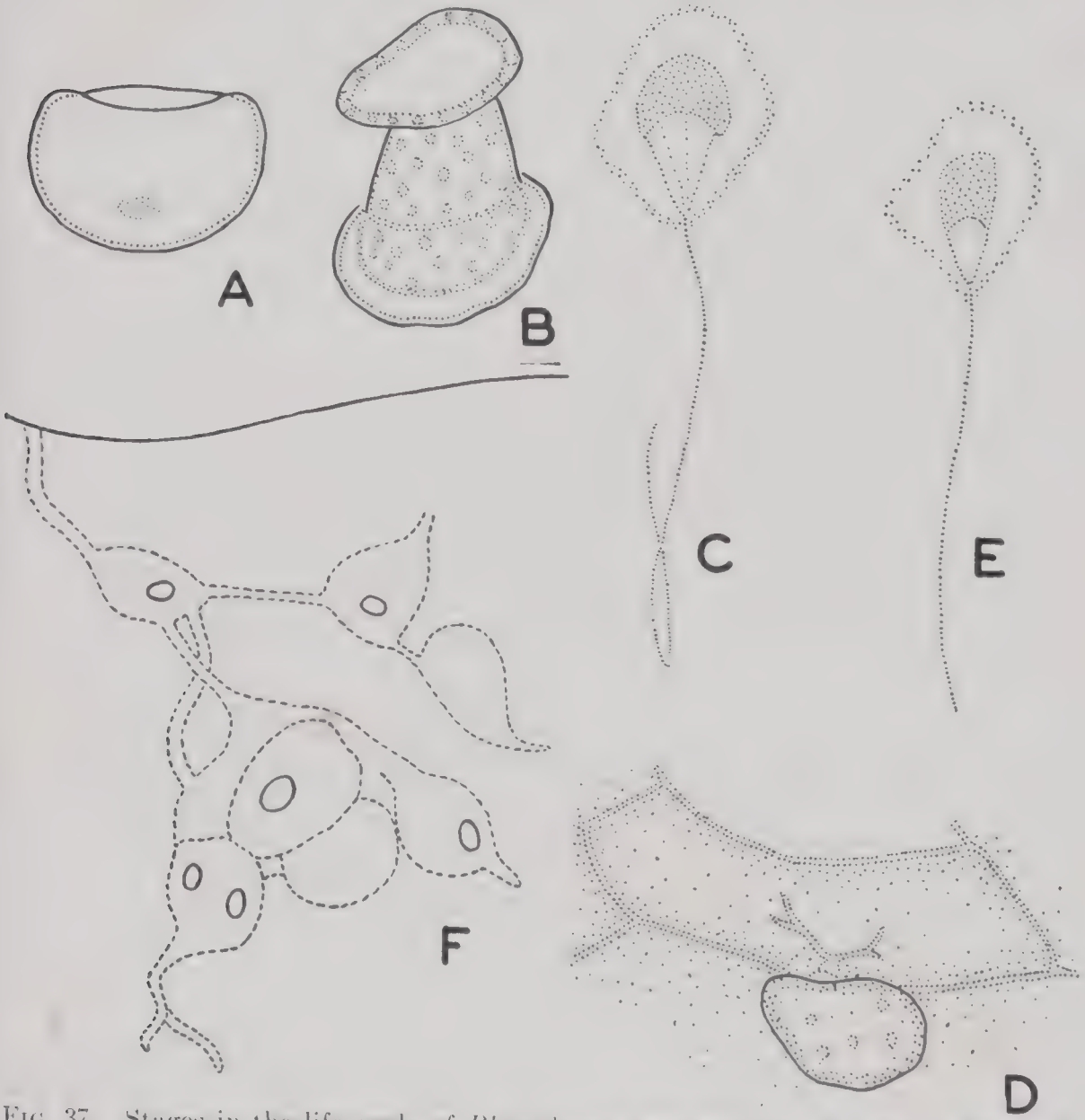

FIG. 37. Stages in the life cycle of *Physoderma zeae-maydis*. *A*, resting spore; *B*, dehisced operculum being carried up by the formation of the sporangium during germination of the resting spore; *C*, zoospore from the sporangium; *D*, ephemeral sporangium; *E*, zoospore from the ephemeral sporangium; *F*, vegetative cells (sammelzellen) in the host tissue. (*A–E after Sparrow; F after Tisdale.*)

turity, through a single apical papilla, 300 or more zoospores, which are much smaller than the zoospores produced in resting spores (2). Presumably these latter zoospores act as gametes, but their union to form a zygote has not been observed. Mycelium in the host cell consists of fine fibrous hyphae which connect large vegetative cells, termed **sammelzel-**

len. The latter may eventually develop into resting spores or send out fibrous hyphae which produce terminal spores.

Disease Cycle. The fungus is an obligate parasite. The resting spores produced in the plant tissue remain viable in dry soil and in leaf sheaths for as long as 3 years. They will germinate, however, at least some of them, as soon as they are mature. They are released from the host tissue as the lesions mature and are readily disseminated by wind. This does not ordinarily take place in time to permit local secondary infection during the current season. The relatively high temperature requirement (23 to 30°C.) for germination of the resting spores is probably an important factor in restricting the disease in severe form to southern states. The fungus is intracellular in the host tissue, and the fibrous hyphae grow from cell to cell. Slight enlargement of invaded host cells is induced, but there is no pronounced hypertrophy and hyperplasia as in potato wart and alfalfa crown wart.

Control. Rotation and sanitation are of limited value because of the widespread dissemination of the resting spores by wind. The disease has not become sufficiently destructive to instigate the development of specific control measures. It appears that hybrid lines of corn differ somewhat in susceptibility, and, if the problem warrants it, control might be accomplished most satisfactorily through development of resistant varieties.

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CROWN WART OF ALFALFA

This disease was first described from Ecuador by Lagerheim in 1895. It was reported from Alsace in 1902, from Italy in 1905, and from Germany in 1909. Later reports in Europe indicated that the disease occurs in England, Holland, and Sweden. The first report in the United States came from California in 1909. Its major distribution in this country has been in Pacific coast areas. It has been reported from Utah and Montana. It occurs on common alfalfa, *Medicago sativa* L., and on *M. falcata* L.

Symptoms. The galls or warts appear on buds which emerge from the crown of the plant, especially on those which arise early in the spring at points below the surface of the soil. The infected buds become slightly thickened and rounded, and as growth progresses, they are glistening white in color. As scale leaf and stipule elements in the bud are affected,

FIG. 38. Crown wart of alfalfa; *gl* indicates the ground level. (*After Jones and Drechsler.*)

they grow at different rates, and the hypertrophied mass takes on an irregular form (Fig. 38). Most of the galls are 2 in. or more below ground. If they protrude above the surface, they take on a green color. They reach full development in early summer, at which time decay begins to set in and a majority of the galls disintegrate. Some of those located deep in the soil become corked over and survive the winter. When galls are

cut open, irregularly shaped brown masses of spores of the causal organism are visible in the interior.

The Causal Organism. *Urophlyctis alfalfae* (Lagerh.) Magn., 1902.

Synonymy: *Cladochytrium alfalfae* Lagerh., 1895.

The yellowish resting spores produced in diseased tissue are the means of the perpetuation of the fungus in the soil. They germinate, according

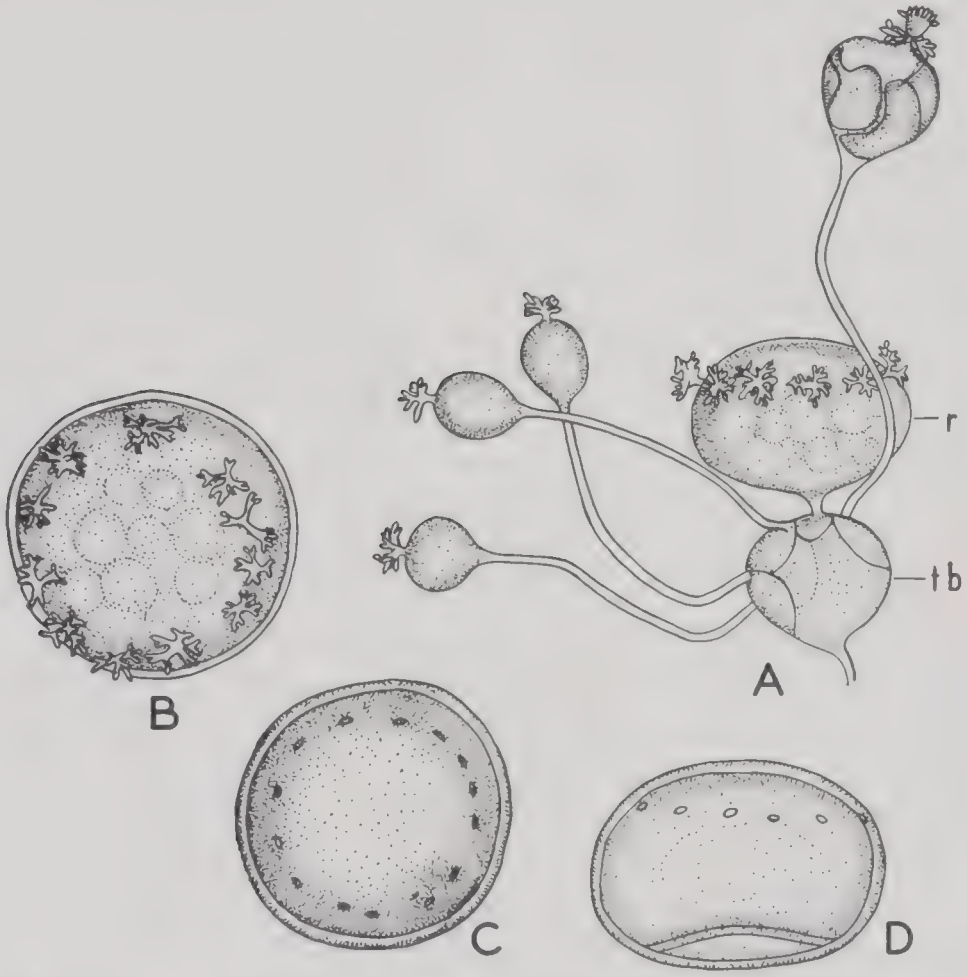

Fig. 39. Stages in the life cycle of the alfalfa wart organism. *A*, the actively growing thallus dissected from the host tissue; *tb*, turbinate cell; *r*, resting spore. *B*, resting spore, showing haustoria in zonate arrangement. *C*, *D*, resting spores, showing pits marking former locations of haustoria. (*After Jones and Drechsler.*)

to Scott (1), by production of 1 to 15 sporangia from each spore. Zoospores escape from the sporangium through a number of tubes in the hyaline wall. Zoospores (3 to 4 μ in diameter) become attached to bud tissue. One to three **sammelzellen,** or **turbinate bodies,** occur in a single epidermal cell of the host, each attached to what was presumably the penetration hypha. The turbinate cell is at first uninucleate, but as it grows, it becomes multinucleate and sends out several branches, each of which in turn produces a turbinate cell. The protoplasm of the older

cell moves into the new cells as it increases in volume, and the old cell eventually becomes segmented and evacuated. At the tip of each turbinate cell a haustorium-like projection is formed, which does not enlarge until evacuation of the turbinate cell has progressed somewhat and walls within the cell have begun to divide it into segments. It then swells, and when it reaches about 5 μ in diameter, 9 to 15 slender, branched haustoria appear in a zone midway between the equator and the distal pole. The body continues to enlarge (25 to 35 by 45 to 50 μ) to form the resting spore, which at full size is several times as large as the turbinate cell to which it is attached. The outer wall becomes quite thick, and probably the haustoria serve as a means of absorbing food from the host cell during growth. As the spore matures, the haustoria disappear, leaving a circle of pits or scars at their points of attachment (Fig. 39).

Disease Cycle. The fungus is perpetuated as resting spores in the soil. The conditions which favor infection and disease development have never been worked out. The fact that the disease occurs in relatively few locations indicates that it is restricted to certain combinations of climate and soil. It is most severe where winters are mild and alfalfa is grown under irrigation. On the other hand, it has been reported in Rocky Mountain areas, where winters may be severe.

Invaded cells enlarge, and there is a slight tendency for adjacent cells to be affected before invasion. The fungus progresses from cell to cell, penetrating the cell wall. There appears to be softening of the latter in advance of the growing turbinate cells, and sometimes, but not always, mechanical pressure is exerted by the fungus. As soon as the fungus has begun to invade the tissue, hyperplasial activity becomes prominent. Invaded cells are disrupted eventually, and definite cavities are formed progressing inward from the invaded epidermal cell. The rapid division of cells around the cavity tends to bury the latter so that it no longer leads to the exterior of the gall.

Control. No control measures have been worked out.

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APHANOMYCES ROOT ROT OF PEA

The root rot of garden pea incited by *Aphanomyces euteiches* Drechs. is one of several diseases of this host which affects the subterranean organs. It was first described fully by Jones and Drechsler (5) in 1925, following studies made chiefly in Wisconsin during the previous 5-year period. It

has since been found in many other states and in Europe and in Australia. In some years in Wisconsin this disease is the most destructive to the crop. Walker and Hare (11), in a critical survey in 1942, concluded that some 10,000 acres were lost in that state primarily from this root rot. The fungus has been found in the roots of hairy vetch, sweet pea, cowpea, sweet clover, alfalfa, and tomato, but serious losses are not caused on any of these hosts. Other important diseases incited by related species are the damping-off and tip rot of sugar beet (*A. cochlioides* Drechs.) (1–3) and black root of radish (*A. raphani* Kendr.) (6).

Symptoms. In the early stages of the disease the tissue of the lower stem and root outside the vascular cylinder becomes soft and water-soaked. As the decay proceeds, fine roots are killed, and when the plant is pulled, the upper taproot separates easily from the rest of the root system, which remains in the soil (Fig. 40). The decay of the stem may extend an inch or two above ground, and decayed tissue of stem and root becomes discolored, chiefly by the invasion of other soil fungi. When plants are grown in pure quartz sand and inoculated, the decayed roots are nearly colorless. The final diagnosis of root rot, because of the complication with other more or less pathogenic soil fungi, is usually made by examination of roots under the microscope for the characteristic relatively thick-walled oospores described below. The stem decay often extends 1 or 2 in. above ground. The leaves of the plant shrivel progressively upward, the top is dwarfed, and in severe cases the plant collapses and dies.

The Causal Organism. *Aphanomyces euteiches* Drechs., 1925. The most conspicuous feature of the fungus upon examination of diseased tissue is the oospore, which is subspherical with a thick colorless wall and varies from 18 to 25 μ in diameter, the wall being about 1.5 μ in thickness. The content of the oospore consists of a large spherical vacuole-like body, of apparently homogeneous structure, surrounded by a granular matrix. The oospore germinates without a required rest period by one to three germ tubes or by a single hyphal filament, which, when it is 200 to 350 μ in length, functions as a sporangium, giving rise to a dozen or more biciliate zoospores (Fig. 41). The mycelial thallus, which arises from direct germination of the oospore or the quiescent zoospores, is hyaline, nonseptate, and with frequent short branches. Under favorable conditions, as observed on culture media, the relatively young branches become sporogenous, serving as sporangia without any discernible morphological distinction as such. The protoplasm becomes segregated within the sporangial filaments into zoospores, which are discharged from the apical tips in very great numbers. While this type of asexual reproduction in this fungus has not actually been seen in nature, its common occurrence

in pure culture is the reason for the belief that it is an important stage in the life history of the organism. As the zoospores leave the sporangial tip, they are cylindrical, but they soon round up into spherical masses, which secrete thin peripheral walls and remain in a clump at the point of discharge. In 1 to 2 hr. the quiescent spore germinates by the formation of a papilla, which expands to a spherical vesicle into which the proto-

FIG. 40. *Aphanomyces* root rot of garden pea. *Left*, healthy plant; *center*, cortical decay of taproot and lower stem; *right*, complete decay of fibrous root system leaving fibrovascular tissue of taproot.

plasmic contents stream rapidly. Two cilia develop, and the zoospore swims away for a variable period, after which it comes to rest and germinates by one to three germ tubes. The phenomenon of there being two motile stages is known as **diplanetism.**

The sexual stage develops readily on culture media and in the host tissue. When this stage is reached, the vegetative phase ceases promptly. Oogonia develop terminally on hyphal branches as thin-walled subglobose

bodies with densely granular vacuolate contents. Antheridia arise from hyphae which usually have no close organic connection with those bearing the oogonium. One to four antheridia become attached to a single oogonium. After fertilization, the oogonial wall becomes conspicuously

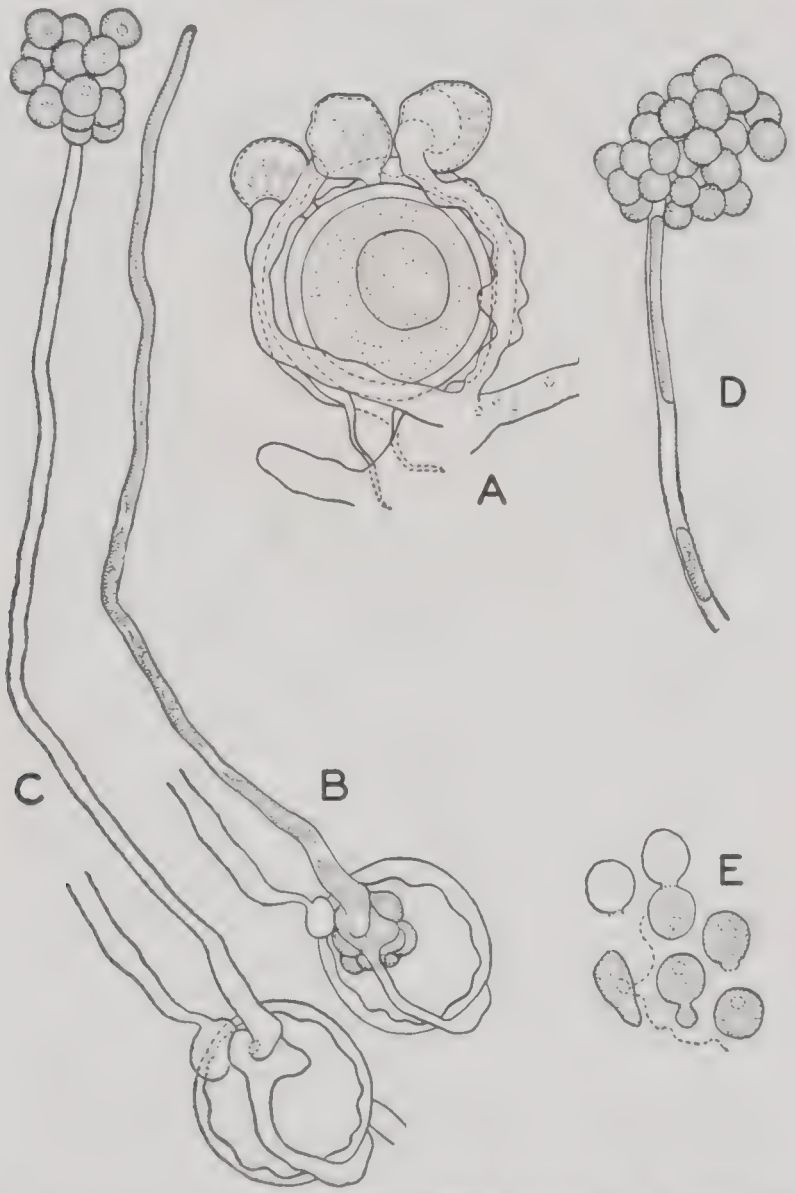

FIG. 41. Stages in the life cycle of *Aphanomyces euteiches*. *A*, oogonium and antheridia in process of fertilization of the former; *B*, germ sporangium arising from germination of oospore 30 sec. before discharge of zoospores; *C*, zoospores after evacuation of sporangium; *D*, evacuation of zoospores from sporangium (asexual reproduction); *E*, evacuation of encysted zoospores and motile stage after evacuation of encysted stage. (*After Jones and Drechsler.*)

thickened, the inner boundary of the wall taking on a scalloped appearance. The oogonial cavity is nearly completely occupied by the mature oospore.

Disease Cycle. The organism remains active in infested soil for many years. Whether this is due to longevity of the resting spores, to the perpetuation of the fungus as a mild parasite on the roots of other hosts,

or to its existence as a free-living saprophyte has not been ascertained. In an extensive survey in Wisconsin in 1942 (11) it was shown that disease incidence declined in proportion to the length of period between pea crops. Infection of the host takes place by direct penetration. The mycelium is intracellular. Infection and disease development occur at soil temperatures of 10 to 30°C. or above, with an optimum about 24°. Soil moisture is extremely influential; in general, dry soil restricts infection while high soil moisture favors it. It has often been observed that increase in soil fertility reduces the severity of the disease (10), and in some regions application of fertilizer has been suggested as a measure of control (4). Smith and Walker (8), in a controlled-nutrient study, showed that the disease decreased in proportion to increase in salt concentration, while raising or lowering the proportion of nitrogen, phosphorus, or potassium had little effect. In general, root rot is more severe in wet seasons and in poorly drained soils. Some soil types, particularly those with high water-retaining capacity, favor development of the disease in comparison with other types (9, 11).

Control. Long rotations are essential. It is important to select soils which are of good texture, not clayey or sticky, and well drained. Reasonably high fertility is helpful. No progress in the development of resistant varieties has been made.

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PYTHIUM DISEASES

Root rots and damping-off are extremely destructive diseases of many plants. They usually affect subterranean portions first and often advance above ground and produce rapid killing. Similar symptoms are commonly incited by quite unrelated organisms. In many cases phycomycetous fungi are involved, as in the cases represented by the species of *Aphanomyces* described in the preceding section. Another group of soil Phycomycetes which are common causal agents of root rot and damping-off is composed of various species of Pythiaceae, of which members of the genera *Pythium* and *Phytophthora* are the most common. Many species of *Pythium* have been described as root-rot pathogens. Some also attack such vegetable fruits as cucumber and melon and vegetative organs, such as cabbage heads and turnip roots. Altogether, species of *Pythium* constitute one of the most destructive groups of plant pathogens.

Symptoms. *Damping-off* is the phase which affects young seedlings. Infection occurs usually just below the soil line on the hypocotyl or upper taproot. The young plants of many of the dicotyledons are expanding rapidly at this time by elongation of cells with relatively little cork cambium and fibrovascular cambium activity. Cell-wall membranes are generally thin, and the tissue is particularly vulnerable. Crowding of seedlings may increase susceptibility since it tends to delay cell-wall thickening and lignification. *Pythium* and other damping-off fungi, which as a class are relatively low grade pathogens, cause upon invasion rapid killing of cells and collapse of the tissue. The damage may be complete before emergence of the seedling above ground, or the disease may continue to develop after emergence. Commonly, plants which are infected after wall thickening and cambium activity get under way, resist or outgrow the disease successfully.

Root rot and *stem rot* are phases which affect many plants after they have become established. The fibrous root system is attacked, and rootlets are softened and killed, the above-ground effects consisting in stunting, sudden wilting, or death. When infection occurs near the soil line on stems in which secondary thickening is well established, cortical lesions develop; these may become sloughed and thus be of minor importance, or they may girdle the stem to cause stunting or death. Extension of brownish lesions to petioles is not uncommon. Examples are to be found in root and stem rots of begonia (43, 44), Persian buttercup (49), oat (53), corn (22, 34), sugar cane (47), geranium (6), and pea (27). The milo disease of sorghum is a root-rot complex in which *P. arrhenomanes* occurs, but the chief causal organism is *Periconia circinata* (Mang.) Sacc. (39). A stalk rot of corn (*Pythium aphanidermatum*) distinct from root

rot has been described (23). The only true foliage blight reported so far that is incited by a species of *Pythium* is the tip blight of pea (*P. ultimum*) described by Hare (27).

Soft rot is the phase which affects the fleshy organs of many plants, particularly the vegetables. Here the pathogen incites decay which is

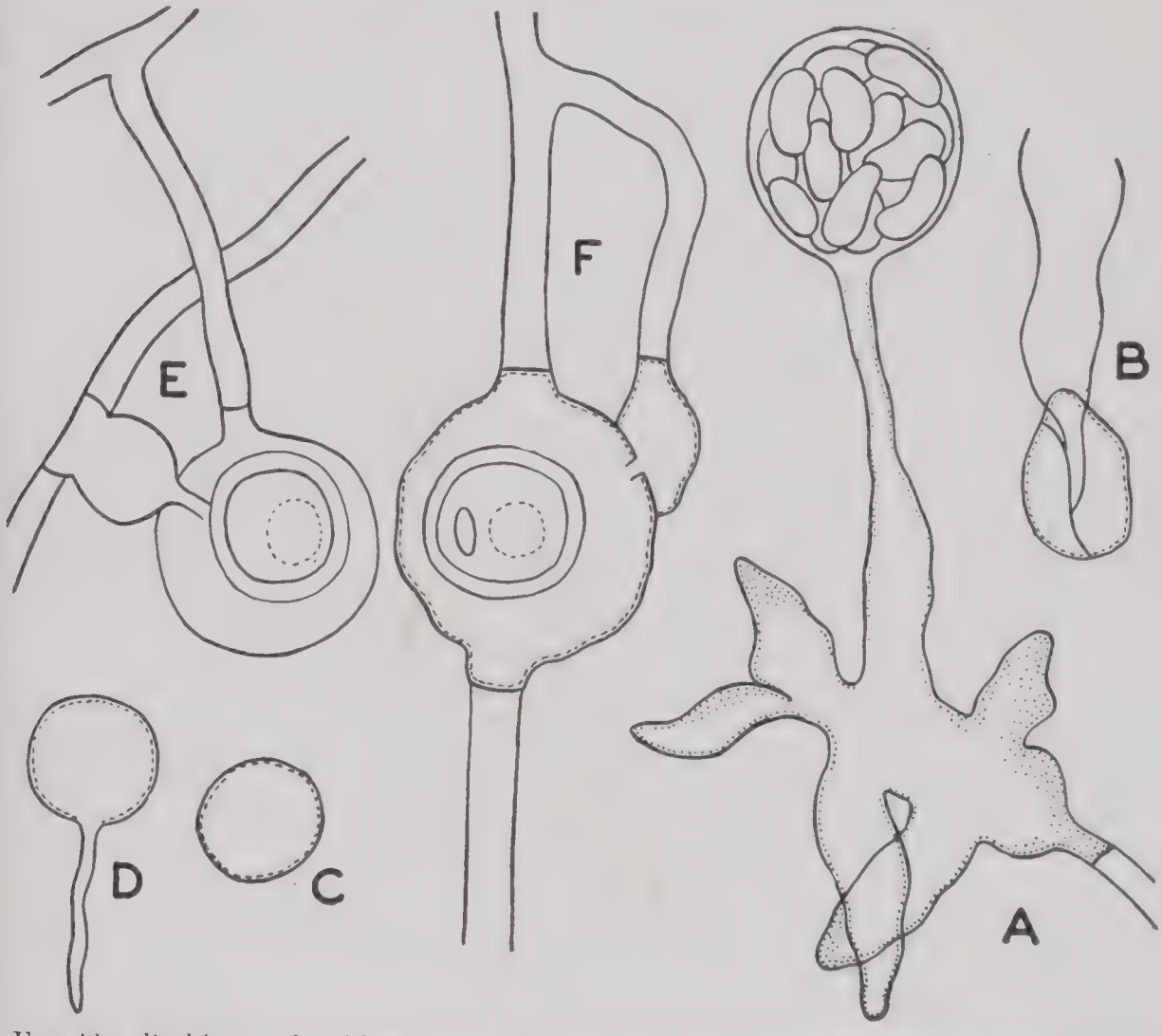

FIG. 42. *Pythium aphanidermatum*. *A*, inflated filamentous sporangium with vesicle containing zoospores; *B*, zoospore; *C*, encysted zoospore; *D*, encysted zoospore producing germ tube; *E*, oogonium with intercalary antheridium; *F*, oogonium with terminal antheridium. (*After Middleton.*)

important in storage, transit, and market. Cucurbit fruits (13, 48), cabbage heads (14), and potato tubers (31) are examples of host substrates which are affected. Infection takes place through wounds or where fruits are in contact with wet soil, and progress is usually rapid. The tissues become water-soaked, lose cohesion, and liquid is exuded, much as in bacterial soft rot. The fungus is intracellular and may be inconspicuous in the early stages of decay, but it eventually produces fluffy or adpressed white aerial mycelium on the surface of decayed tissue.

Mycelium which develops outside the substrate is referred to as **extramatrical.**

The Causal Organisms. A large number of distinct species of *Pythium* have been described. Comprehensive monographs of the genus are those by Butler (10), Matthews (41) and Middleton (45). While most species can ordinarily be distinguished by microscopical study, there is relatively little specificity with regard to host plant or phase of disease.

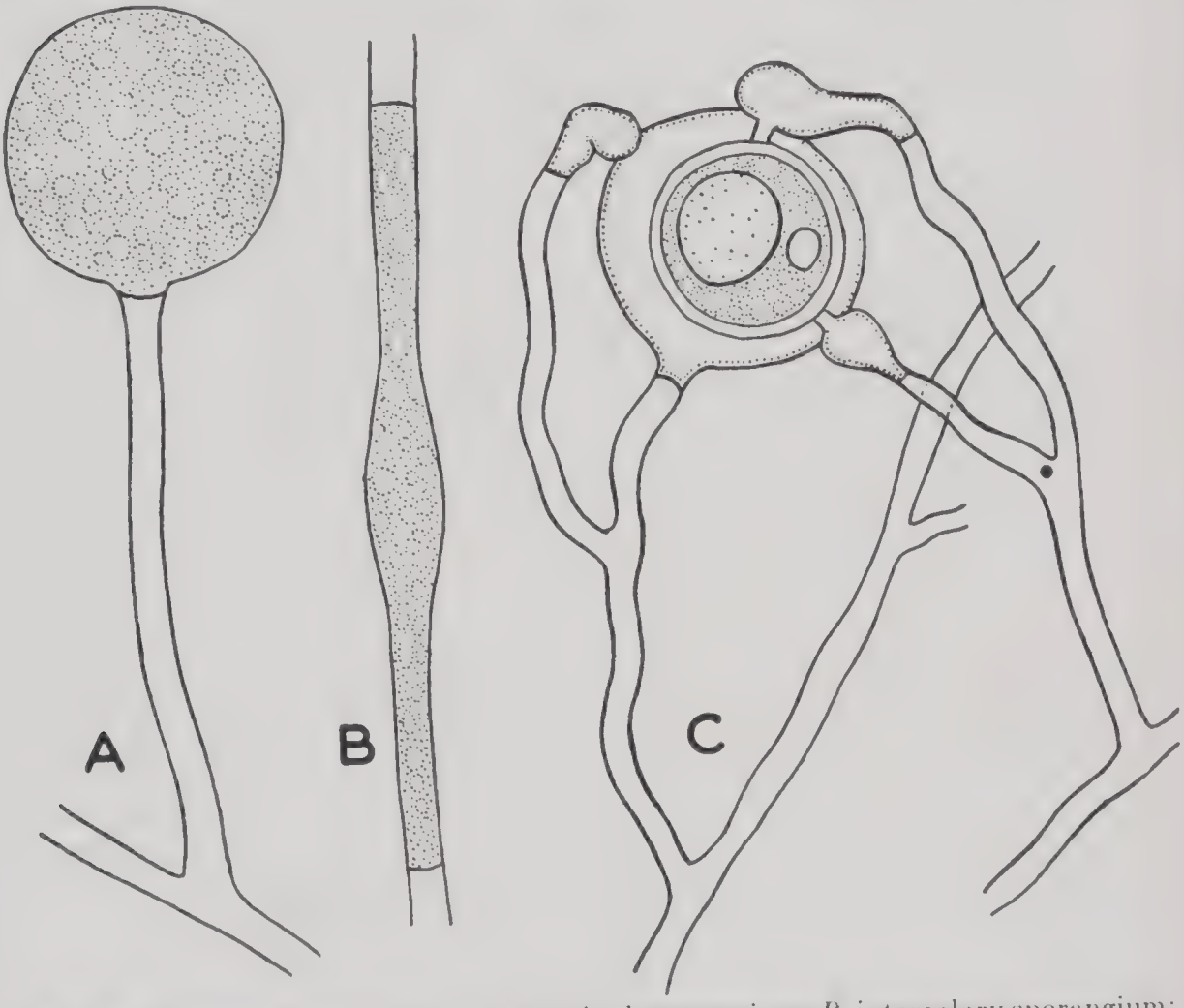

FIG. 43. *Pythium debaryanum*. *A*, terminal sporangium; *B*, intercalary sporangium; *C*, oogonium and antheridia and formation of oospore following fertilization. (*After Middleton.*)

Species which have such a wide host range are called **omnivorous.** Moreover, several distinct species may incite the same disease symptoms on a given host. It is common, therefore, to find a given disease phase incited by one species in one instance and by another species in another case. In infected plant tissue the presence of *Pythium* is often recognized by the occurrence of oospores, but it is important to emphasize that oospores of other Phycomycetes, particularly those of *Aphanomyces* and of *Phytophthora*, may be confused with those of *Pythium*.

The asexual fruiting body is a sporangium, from which the protoplasm passes characteristically through an emission tube in an undifferentiated state to form a vesicle in which zoospores are formed, the process usually taking place in less than an hour. Depending upon the species, the sporangium may be filamentous (Fig. 42) and scarcely distinguishable from the mycelium, it may appear as more or less inflated mycelium, or it may be distinctly spheroidal in form (Fig. 43). In some of the species with spheroidal sporangia, the latter germinate by production of one to six germ tubes, *e.g.*, *P. ultimum*. When zoospores are formed, they number up to 125 per sporangium, are biciliate, and germinate after encystment by a germ tube. In some species a small vesicle may be emitted

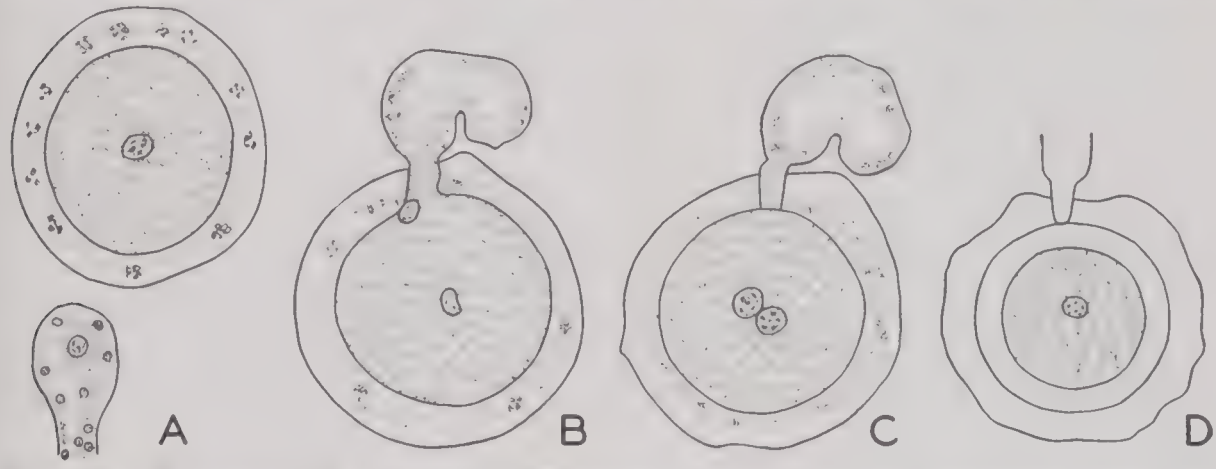

FIG. 44. Formation of oospore of *Pythium debaryanum*. *A*, oosphere with degenerating nuclei in the periplasm; antheridium with one functional nucleus and several degenerating nuclei; *B*, antheridium discharging male nucleus through the fertilization tube; *C*, male and female nuclei in contact; *D*, nearly mature oospore with the oogonial wall still present and the emptied antheridium still attached. (*After Miyake.*)

from the tube, several secondary zoospores formed, and the process repeated several times.

Oogonia are spherical, smooth-walled in some species and echinulate in others. Antheridia vary between species in shape and in origin. Some form from the oogonial stalk at its apex (**hypogynous**); some anywhere along the oogonial stalk (**monoclinous**) (Fig. 42); some from another branch of the thallus (**diclinous**) (Fig. 43). The number of antheridia per oogonium varies. Usually one oospore is formed per oogonium, occasionally two or three. The walls are smooth or reticulate, thin- or thick-walled, and either fill the oogonial cavity (**plerotic**) or only partially fill it (**aplerotic**). Oospores germinate in some cases by formation of hyphae and in others by discharge of contents through an evacuation tube to form a vesicle in which zoospores are formed as in sporangia. The cytological details of oospore formation and maturation are given by Trow (50) for *P. ultimum* and for *P. debaryanum* (Fig. 44) by Miyake (46).

Among the most common species of *Pythium* are *P. arrhenomanes* Drechs., *P. aphanidermatum* (Edson) Fitz., *P. ultimum* Trow, and *P. debaryanum* Hesse. These may be characterized briefly as follows:

P. arrhenomanes. Sporangia inflated, filamentous, lobulate; zoospores about 12 μ in diameter on encystment; oogonia subspherical to spherical, usually acrogenous, smooth, thin-walled, 17 to 56 μ; antheridia diclinous, 15 to 25 per oogonium; oospores plerotic, 15 to 54 μ. Most aggressive species associated with root rots of corn, wheat, and sugar cane, but reported from roots of many species.

P. aphanidermatum. Sporangia inflated filamentous, branched or unbranched; zoospores 7.5 by 12 μ; oogonia spherical, terminal, 22 to 27 μ; antheridia monoclinous or diclinous, intercalary or terminal, one or two per oogonium, dome-shaped, 9 to 11 by 10 to 14 μ; oospores aplerotic, single, 17 to 19 μ. Associated with damping-off and with soft rot of storage organs of many host species (Fig. 42).

P. ultimum. Sporangia mostly terminal and spherical, 12 to 28 μ, germinating only by germ tubes; oogonia smooth, spherical, mostly terminal, 19 to 23 μ; antheridia monoclinous arising just below the oogonium, usually one per oogonium; oospores aplerotic, single, spherical, 14 to 18 μ; smooth- and thick-walled, germinating by a germ tube which at low temperatures emits a vesicle in which zoospores are formed. Common causal organism of damping-off, root rot, and soft rot of many species and of tip blight of garden-pea foliage (Fig. 45).

P. debaryanum. Sporangia spherical to oval, terminal or intercalary, 15 to 26 μ, germinating by germ tubes or zoospores; oogonia smooth, terminal or intercalary, usually spherical, 15 to 28 μ; antheridia monoclinous or diclinous, one to six per oogonium; oospores smooth, aplerotic, 12 to 20 μ, germinating by germ tubes which at low temperatures give rise to vesicles in which zoospores are formed. Common causal organism of damping-off, root rot, and soft rot of many species (Figs. 43, 44).

Disease Cycle. Species of *Pythium* are natural inhabitants of soil, where they probably subsist as saprophytes and often as low-grade parasites on fibrous roots. The oospores serve as overwintering organs. In most species the optimum for growth on a favorable agar medium is about 28°C. or slightly above. Compared with those of many fungi, the mycelium grows very rapidly.

The influence of soil environment on disease development has been studied with a number of diseases. Johann *et al.* (34) found that preemergence damping-off of corn incited by *P. arrhenomanes* was enhanced by low temperature and high moisture. Reduction of postemergence plant height and dry weight was favored by the same conditions. In browning root rot of wheat, incited chiefly by the same species, Vanterpool

and Truscott (52) also found high moisture favorable. Preemergence killing was severe at 12°, 17°, and 31° but less at 24°. Postemergence killing, however, was least at 12° and increased with temperature up to 31°. In this phase the temperature effect in wheat was diametrically opposite that in corn. Rands and Dopp (47) reported that root rot of sugar cane incited by the same species was most severe at 19° and became less severe with increase in temperature up to 33°. Hemmi (32) noted that postemergence damping-off of garden cress, incited by *P. debaryanum*, was more severe at soil temperatures of 20 to 28° than at lower temperatures. Greeves and Muskett (26) found that in the case of rutabaga seedlings affected with *P. debaryanum* preemergence damping-off was most severe at low temperature (average 5.8°), while postemergence damping off was favored by high temperature (average 23.0°). It is evident that the effect of soil factors on host growth had a definite bearing on disease development. In the warm-weather hosts, corn and sugar cane, the postemergence phase of the disease declined with increase in temperature, while in the cool-weather crops, wheat, cress, and rutabaga, the opposite relation held.

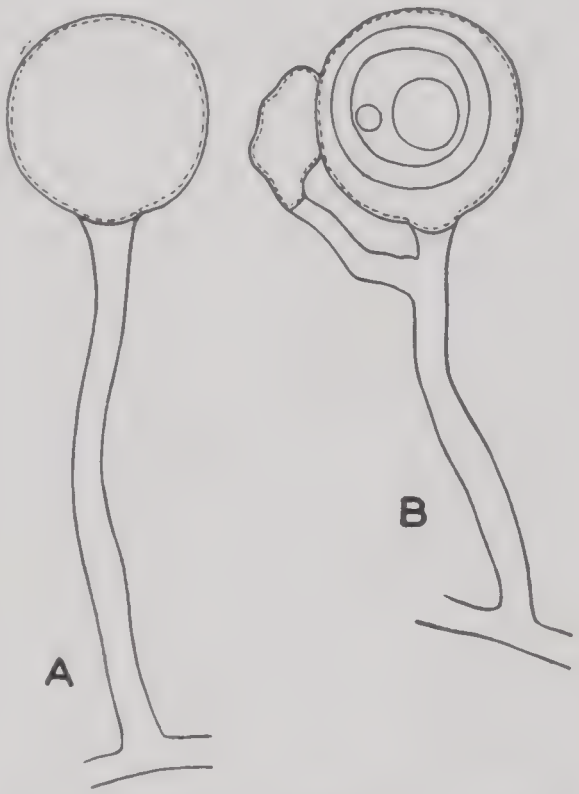

FIG. 45. *Pythium ultimum*. *A*, acrogenous sporangium; *B*, oogonium, antheridium, and oospore. (*After Middleton.*)

In a study of preemergence damping-off incited by *P. ultimum*, Leach (38) considered the rates of growth of the pathogen and of several hosts. The "coefficient of velocity," *i.e.*, the ratio of the rate of emergence of the host to the rate of growth of the fungus, was determined. This ratio was found to be correlated closely with the rate of preemergence damping-off of a given host at different soil temperatures. When the ratio was below unity at a given temperature, the host was growing more slowly than the pathogen and damping-off was heavy; as the ratio increased beyond unity, the disease declined accordingly. This accounts in large measure for the fact that in high-temperature vegetable crops, such as lima bean, watermelon, and muskmelon, preemergence damping-off is most severe at low temperatures. In such low-temperature crops as spinach and pea more damage is encountered at intermediate and high soil temperatures.

Drechsler (19) calls attention to the fact that oospores of *P. ultimum* and *P. debaryanum* germinate to form zoospores more readily at low than at high temperatures. He suggests that this may account for the excessive blackening of root tips by these species at low soil temperatures.

The browning root rot of cereals (*P. arrhenomanes*) is sometimes severe in midwestern Canada, particularly following summer fallow. Vanterpool (51) showed a close correlation of disease severity with low phosphorus and high nitrate nitrogen content of the soil. Carpenter (11, 12) claimed that sugar cane become more susceptible to *Pythium* root rot when supplied with excessive amounts of nitrogenous fertilizer. McClure and Robbins (40), working with *P. splendens* on young cucumber seedlings, found the plants more susceptible in low as compared with normal light intensity and in low nitrogen as compared with balanced nutrition. There was a correlation between lignification of cell walls and resistance of the plant to attack.

In the decay of storage organs the reactivity of the host tissue may be less important in the disease complex. Goss and Jensen (25) noted that rot of potato tubers incited by *P. debaryanum* was most rapid at 25°. Harter and Whitney (29) found that in *Pythium* rot of sweet-potato roots (*P. ultimum* and *P. scleroteichum* Drechs.) the type of decay varied with temperature. From 2 to about 18° a cheesy type of rot was produced, while above 18° the tissue became more crumbly and mealy as temperature increased. The optimum for decay of snap beans by *P. aphanidermatum* was 31° (37).

Control. Preemergence damping-off is often well controlled by treating seeds with a suitable protectant. This is discussed in Chap. 16. Decay of potato seed tubers after planting was reduced by applying a dust protectant to cut seed or allowing callus formation by keeping the cut pieces in a humid atmosphere 48 hr. before planting (35).

Root rot is not so easily prevented by direct means, although provision of good soil drainage is sometimes helpful.

In some cases development of resistant varieties is feasible. The need and opportunity for this approach in sugar cane is discussed by Rands and Dopp (47). The occurrence of resistance to *Pythium* root rot in certain lines of corn is reported by Johann *et al.* (34) and by Elliott (22). The root rot of sorghum has been successfully controlled by development of resistant varieties (5, 42), but whereas the chief causal organism was regarded as *P. arrhenomanes* when those studies were conducted, Leukel (39) has since shown that the resistant character applies to *Periconia circinata*, which is now regarded as the major pathogen. Hawkins and Harvey (31) showed that the resistance to soft rot (*P. debaryanum*) in some varieties of potato was due to the mechanical barrier provided by

the host-cell walls against penetration. No commercially acceptable *Pythium*-resistant potato varieties have been developed, however.

Control of soft rot of storage organs is reduced chiefly by careful handling to avoid bruising.

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PHYTOPHTHORA DISEASES

The most important and best-known *Phytophthora* disease is late blight of potato and tomato, treated in the next section. This is a foliage blight and a rot of fruits and tubers. Some other species of *Phytophthora* are soil fungi which are pathogenic on roots and lower stems and on storage organs, inciting diseases which are much like the *Pythium* diseases. A few species of *Phytophthora* have restricted host ranges (*e.g.*, *P. phaseoli* Thaxter, incitant of downy mildew of lima bean (3), and *P. fragariae* Hickman, incitant of the red stele root disease of strawberry (1, 8). Others are omnivorous, several causing quite similar diseases on the same host and some infecting a very extensive list of host species (23). The most complete monograph of the genus is by Tucker (22). Some of the better-known *Phytophthora* diseases, in addition to those mentioned, and their pathogens are listed below:

Crown rot of rhubarb, *P. parasitica* Dast. (7).
Crown rot of hollyhock, *P. megasperma* Drechs. (5).
Fruit rot of pear and apple, *P. cactorum* (Leb. & Cohn) Schroet. (15).
Brown rot of lemon, *P. citrophthora* (R. E. Sm. & E. H. Sm.) Leonian (19).
Pink rot of potato, *P. erythroseptica* Pethyb. (12).
Tomato root rot, *P. cryptogea* Pethyb. & Laff.; *P. parasitica*.
Buckeye rot of tomato, *P. parasitica*; *P. capsici* Leonian; *P. drechsleri* Tucker, (17, 18).
Root rot and fruit rot of pepper, *P. capsici* (10).
Coconut bud rot, *P. palmivora* Butl. (13).
Lilac blight, *P. syringae* Kleb.; *P. cactorum* (2).
White tip of leek, *P. porri* Foister (6).
Black shank of tobacco, *P. parasitica* var. *nicotianae* Tucker (20).
Root rot of cauliflower, *P. megasperma* (21).
Root rot of ginseng, *P. cactorum* (16).
Foot rot of lily, *P. cactorum* (4).

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LATE BLIGHT OF POTATO AND TOMATO

Although the potato was introduced into Europe from South America in the sixteenth century its great expansion as a food crop there and in North America did not come until some 200 years later. Likewise, the tomato, taken to Europe from South America, did not become widely used until after 1800. Late blight appeared almost simultaneously in Europe and in the United States, records beginning about 1830. In succeeding years it increased in extent and severity, culminating in a widespread epidemic on the European continent in 1845, when it contributed greatly to the famine in Ireland. It has continued to be a major disease in the cool, humid regions of the temperate zones. In Europe, the countries surrounding the North Sea are most regularly affected, while in the United States some of the most favorable areas for disease development are located in New England, New York, and Pennsylvania. Epidemics commonly cover a much wider range, and winter-grown crops in the southern states are now regularly affected to some degree. While the crops of semiarid states have been usually free from late blight, spasmodic occurrence is sometimes extremely destructive under such circumstances because of lack of preparedness for the application of adequate control measures. It was reported on potato for the first time in Florida in 1903, in California in 1904, in Manitoba in 1928, in Texas in 1931, in Kansas in 1936, and in Colorado in 1941.

The disease is sometimes extremely destructive to tomato, but the epidemics on this host are not always coincident with those on potato. It was first recorded on tomato in France by Tulasne in 1854. An extensive epidemic in eastern United States in 1946 caused losses running into millions of dollars. The disease has been recorded on eggplant but not upon pepper. There is an extensive host range within the Solanaceae.

Symptoms. The disease may appear on the tops of tomato and potato at any time during their development, favorable environment being a prime conditioning factor. On leaflets, rachis, petiole, or stem the first signs consist in brownish- to purplish-black lesions, which are not delimited in size and under favorable conditions enlarge rapidly (Fig. 46). Lesions may appear near the ground and on lower sides of leaflets first, if humidity and temperature are marginal, but such localization is not usual when the environment is continuously optimal. Under the latter set of conditions a rapid general blighting of foliage occurs. When lesions on individual leaflets are examined, a zone outside the purplish lesion is found to show a paler than normal green merging into the latter, while on the lower side of the leaf a white mildew appears on the surface of the lesion in the region

where the pale and purplish tissues join. This mildew consists of the sporangiophores and sporangia of the pathogen. If the weather becomes clear and the humidity low, the progress of the disease is checked, the pale zone becomes less pronounced, and the mildew disappears. With return of favorable weather the fungus becomes active, and corresponding symptoms and progress reappear. When the blight is advancing rapidly in a potato field, a mildly pungent characteristic odor is given off, which to

FIG. 46. Late blight of potato.

experienced growers is a harbinger of the disease ahead of the conspicuous blight phase.

Potato tubers are often infected while in the hill, and they are subjected to infection at harvest and sometimes in storage. The first sign is a brown to purple discoloration of the skin followed by a brownish dry rot which extends to about ½ in. below the surface. When the disease proceeds without complication, the tuber symptoms are quite distinctive. In storage bins, however, the "wet rot" phase sets in unless ideally cool, dry conditions prevail. The slimy soft decay, or wet rot, is generally the effect of secondary bacteria which follow late blight development. In

moist atmosphere white tufts of mycelium and sporangiophores of the fungus appear on the surface of the tuber.

On tomato fruits symptoms appear at any stage of growth and may be most frequent near the stem end. Gray-green water-soaked spots enlarge to indefinite size and shape (Fig. 47). In transit the rot may be soft and watery. On green fruits in the field the spots assume a dark brown color with a firm wrinkled consistency and a fairly definite border, the latter remaining green as the unaffected tissue ripens to normal color. The fungus may fruit on the surface if the environment is humid.

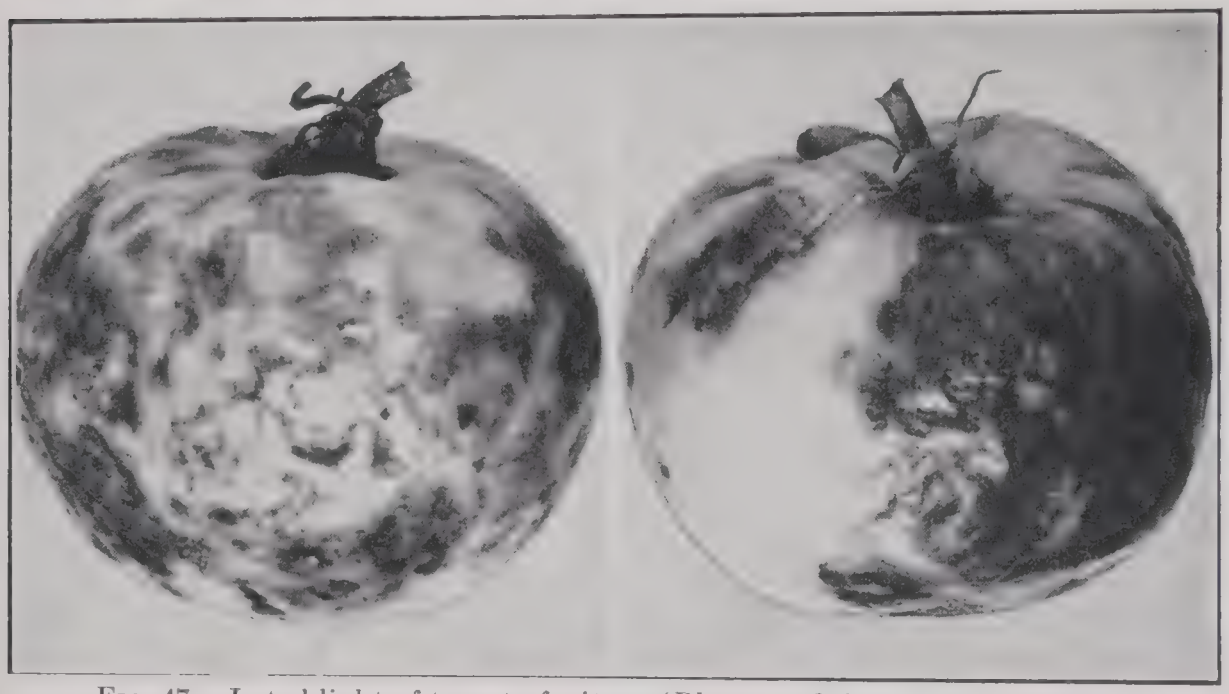

FIG. 47. Late blight of tomato fruits. (*Photograph from R. H. Larson.*)

The Causal Organism. *Phytophthora infestans* (Mont.) DBy., 1876.

SYNONYMY:

Gangraena tuberum solani Mart., 1842.
Botrytis devastatrix Lib., 1845.
Botrytis infestans Mont., 1845.
Botrytis fallax Desm., 1845.
Botrytis solani Hartig, 1846.
Peronospora trifurcata Ung., 1847.
Peronospora fintelmani Casp., 1852.
Peronospora devastatrix (Lib.) Casp., 1855.
Peronospora infestans (Mont.) DBy., 1863.

The sporangiophores arise from the leaf substrate through stomata and from tuber substrate through lenticels; they are hyaline, branched, and

indeterminate. The thin-walled, oval, hyaline sporangium (21 to 38 by 12 to 23 μ) with an apical papilla is borne at the tip of a branch, and as it approaches maturity, the branch tip swells slightly, proliferates, and turns the attached sporangium to the side as elongation of the sporangiophore proceeds (Fig. 48). A fruiting hypha, then, is characterized by periodic swellings indicating the points at which sporulation has taken place. This type of fruiting branch is characteristic of the genus *Phytophthora* and serves to distinguish it from the most closely related genus, *Pythium*. The sporangium may germinate directly by means of a germ tube, which may in turn give rise to a terminal secondary sporangium, but most commonly the contents of the sporangium cleave to form about eight biciliate zoospores which emerge in a group and swim away. After a few minutes the zoospores come to rest and germinate by germ tubes, which penetrate through the stomata or directly through the epidermis. The mycelium in the tissue is coenocytic, intracellular and intercellular. Rudimentary haustoria are found in foliage cells, but in potato-tuber

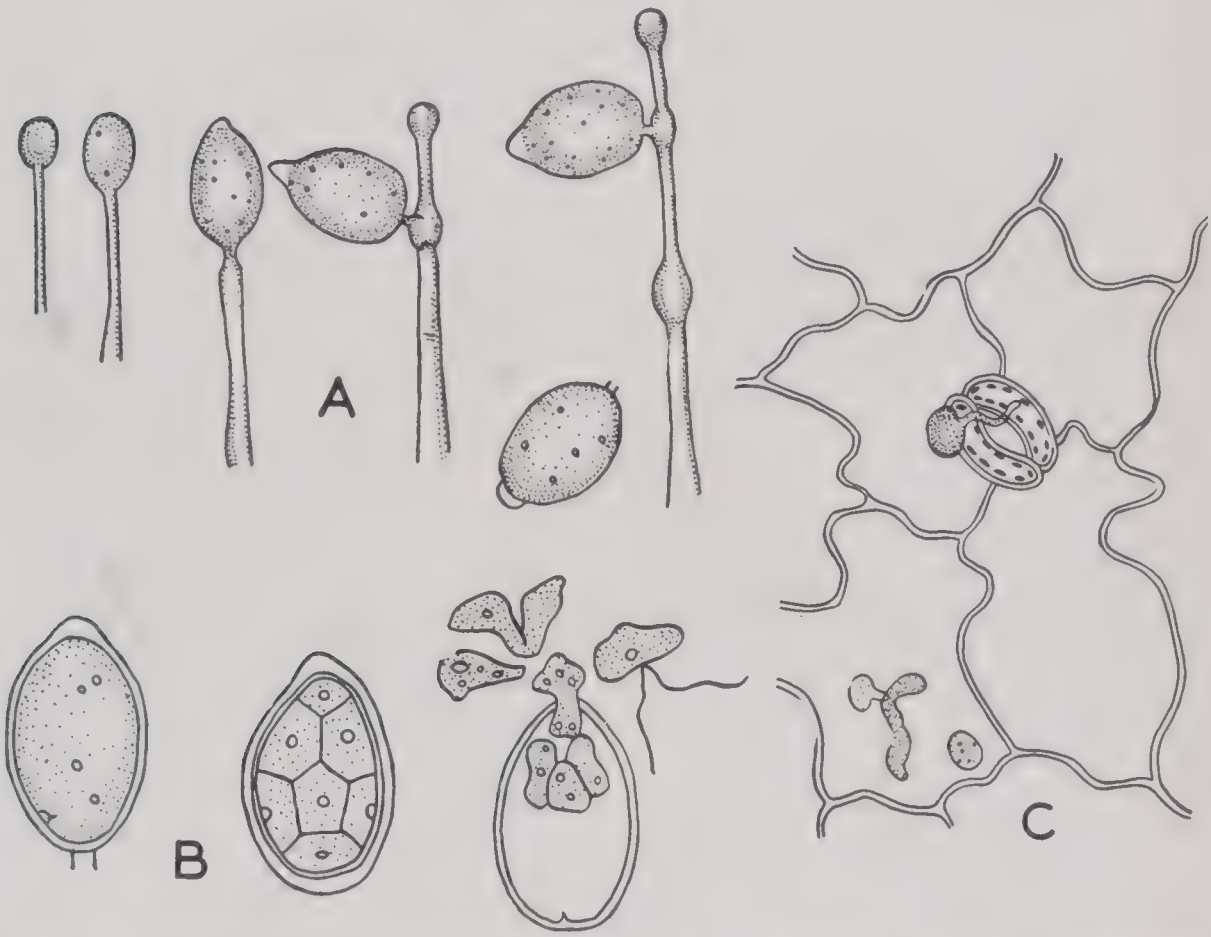

FIG. 48. *Phytophthora infestans*. *A*, formation of the sporangium; *B*, formation of the zoospores; *C*, direct penetration and penetration through stoma by germ tubes from encysted zoospores. (*After Ward.*)

cells they are more elaborate, being club-shaped, hooked, or spirally twisted.

Much controversy arose in the 1870's concerning the overwintering stage of this organism. Since species of the related genera, *Pythium* and *Peronospora*, were known to form oospores, it was natural that investigators looked for and expected to find oospores of the late-blight fungus. These were first claimed by Worthington G. Smith of England. De Bary, however, disputed this claim and showed conclusively that the oospores of Smith did not belong to the late-blight fungus (16). De Bary failed to find any sexual spores, and his claims were substantiated generally by subsequent workers, until the report of Clinton (11) in 1910, who described oospores in pure cultures of *P. infestans*. The genus, *Phytophthora*, had thus been created on the basis of a type species in which sexual spores were unknown. Other species were subsequently described, however, with the characteristic type of sporangiophore and with oospores derived from oogonia, fertilized by antheridia, in the manner found with species of *Pythium*. The oospores of Clinton conform to the type expected. Others have confirmed Clinton's findings. But it is evident that only occasional isolates produce oospores even in culture. The first report of oospores of this species in nature was made by Murphy (31) in potato tubers in Ireland in 1927, but apparently they are exceedingly rare. The induction of oospore formation by pairing of isolates has not clearly indicated heterothallism in this species, but Clinton (11) and, more recently, Barrett (3) have reported that oospores were induced when isolates of *P. infestans* were mated with strains of another species. Oogonia vary from 31 to 50 μ in diameter; oospores, from 25 to 35 μ.

Oospore-like spores (aboospores), which show no evidence of having been formed through a fertilization process, are found commonly in pure cultures. Germination of oospores of *P. infestans* has not been observed. Occasional observations in other species indicate that they germinate by a germ tube. The cytological details of oospore formation and maturation in *P. erythroseptica* are given by Murphy (30).

Disease Cycle. De Bary failed to find overwintering spores of *P. infestans*, and little evidence has accumulated since his time to indicate this means of carry-over as a common one. He showed that the mycelium in infected tubers was the chief means of overwintering. It functioned by invading the shoots of the germinating seed tuber in the spring, emerging above ground with them and sporulating to form the primary inoculum (Fig. 49). Some investigators failed to confirm De Bary on this point but others have, and it is now regarded as the chief means of overwintering. The most convincing confirmatory evidence is provided by Melhus (26). Tubers which overwinter in the soil may be a source of the fungus, as well

as those dumped from warehouses in the spring. In some intensive potato-growing areas this last method has been emphasized as the chief means of overwintering (8). DeBruyn (18, 19) expressed the opinion, based on her studies of the organism in soil cultures, that the fungus overwinters as a saprophyte in the soil in Holland.

Sporangia, once formed, are readily detached and readily air-borne. The conditions which favor spore production, dissemination, and infec-

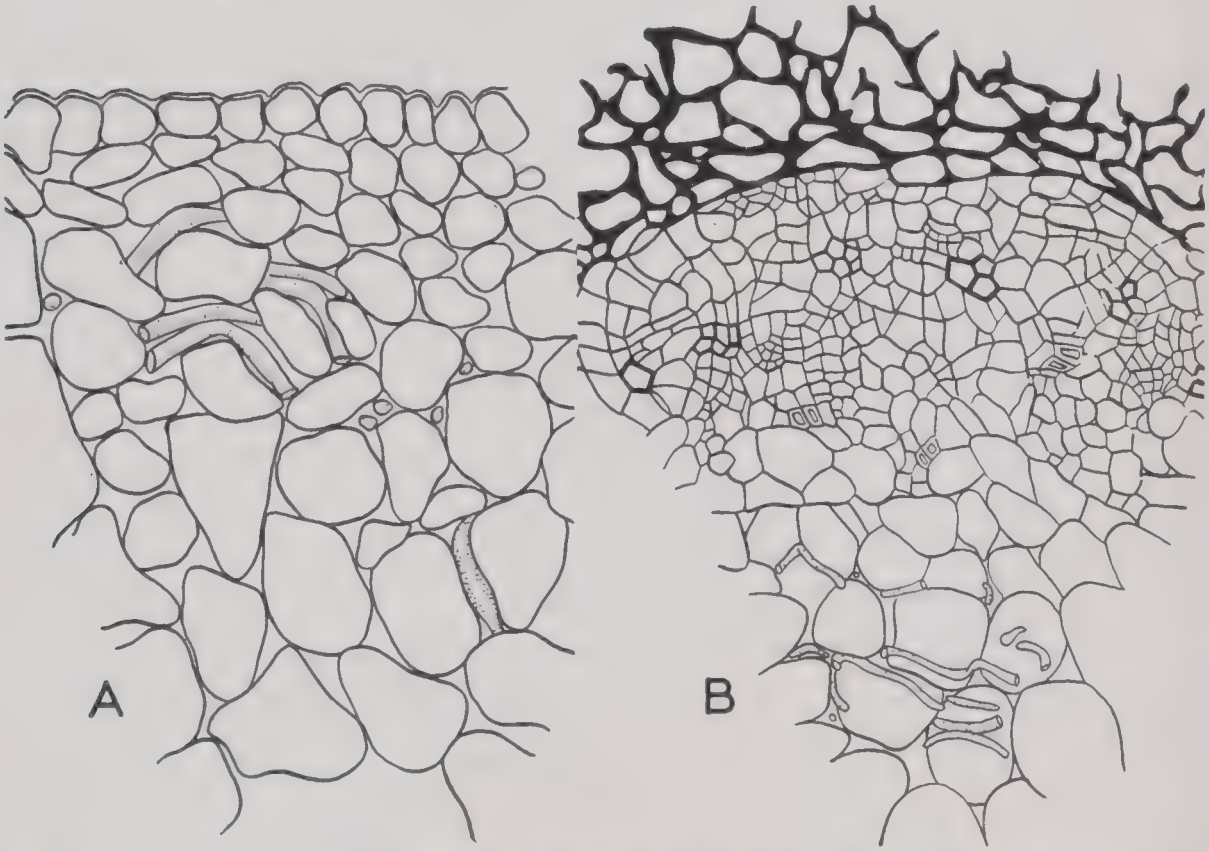

FIG. 49. *A*, mycelium of *Phytophthora infestans* growing from infected tuber in cortex of stem shoot arising from latter; *B*, a later stage in which cortical cells have been killed and the mycelium has invaded the pith. (*After Melhus.*)

tion are those which determine the extent of the epidemic. They have received attention by numerous investigators, among whom Crosier (13) provides very complete data. Sporangia are formed at a minimum relative humidity of 91 per cent with an optimum of 100 per cent, and a temperature range of 3 to 26°C. with an optimum of 18 to 22°. The sporangia formed at 15° reach a peak of zoospore formation at a favorable temperature in 1 to 2 hr.; those formed at 25° require 5 to 7 hr. to reach this peak. The optimum temperature for formation of zoospores is 12°, for formation of germ tubes from sporangia, 25°. The zoospores germinate most rapidly at 12 to 15°. After germination, the germ tubes from zoospores grow best at 21 to 24°. Cool, moist nights are thus required

to provide the most rapid build-up of inoculum and are most favorable for zoospore formation and germination. Slightly higher temperature favors growth after infection. Protracted cold weather may be unfavorable. The disease is of little importance in northern Norway on this account. Thomas (38) has shown that the microclimate in the foliar sphere is more important than the climate 5 ft above the ground in relation to epidemiology.

In some regions where this disease is extremely important, considerable success has been had in forecasting late-blight epidemics on the basis of the above-mentioned data and upon current weather trends. This has been successful in Holland, southeastern England, middle western United States, and eastern Virginia. In Holland and England the weather is seldom too warm to discourage blight, but it may be too cool. The four criteria necessary for late blight are (*a*) night temperature below the dew point for 4 hr. or more; (*b*) night temperature not below 10°C.; (*c*) mean cloudiness not below 0.8 on the following day; (*d*) rainfall of at least 0.1 mm. the following day. In middle western United States the common tendency is for the temperature in the critical months of July and August to be too high for late blight. The major criterion in that area is the duration of periods below 21°. In the Atlantic seaboard area of eastern Virginia a forecast of late blight is made when rainfall exceeds a critical line and the average daily temperature remains below 24° for two successive weeks (12).

Speerschneider demonstrated by experiment in 1857 that the same fungus which caused foliage blight also caused tuber rot. This was not confirmed immediately and was doubted by some, but De Bary (15) demonstrated it beyond doubt by 1863. De Bary (16) claimed that sporangia were carried by water through the soil to the tubers which were penetrated; downward growth through the stem was not a factor. This interpretation has been generally accepted by later workers, but there is little or no experimental evidence to show whether sporangia, zoospores, or mycelium are the means by which the fungus reaches the tubers. Jones *et al.* (23) showed that the percentage of rotted tubers was in inverse proportion to the depth of the soil above them. It has been shown repeatedly that the extent of infection is greatly enhanced if tubers are harvested when foliage is not completely killed and is, therefore, the source of inoculum from fruiting lesions. In fact, the danger of tuber infection at harvest is greater when foliage is kept green by spraying but is still harboring some fruiting lesions than when foliage, poorly protected, has been killed rapidly a week or more in advance of harvest. These factors are taken into account in the control measures discussed under a later heading.

Varietal Susceptibility and Variability of the Pathogen. The development of late-blight-resistant varieties of potato has been going on for nearly a century. Following the impact of the epidemics of the 1840's, attention was given in both Europe and America to the introduction of potato stocks from South America for breeding purposes. One of the most successful breeders of that era was Chauncey Goodrich of Utica, New York, who, in 1851, obtained a variety from South America named Rough Purple Chile. From it were derived Early Rose and Beauty of Hebron varieties. In 1877, Charles Darwin became interested in the possibility of breeding for late-blight resistance and gave encouragement and some financial aid to private breeders in England and Ireland. In 1876, there had been introduced in England a new variety, Magnum Bonum, derived from a cross between Goodrich's Early Rose and a local variety, Victoria. Magnum Bonum had marked tolerance to late blight and remained in high favor until about 1890, when its resistance appeared to decline. Breeding has continued in several European countries since that time, and much progress has been made. All varieties within the species *Solanum tuberosum* L. are susceptible, but varieties differ in the degree of susceptibility. Numerous varieties have been developed by hybridization and selection which have marked resistance to late blight, but in the main they need some fungicidal protection under severe epidemic conditions. This type of resistance is a recessive character and is probably the result of the interaction of several genes (37). Since varieties of potato are allopolyploids ($2n = 48$), inheritance of such a character is complex. That the genes for this type of resistance are in heterozygous condition is shown by the fact that resistant F_1's have been secured from crosses between susceptible parents (*e.g.*, Sebago variety). Resistance of the tops is not necessarily correlated with resistance of the tubers, and these characters may be conditioned by different genes (7). In some cases tuber resistance appears to be due to skin characteristics, in others to characters of the flesh. Among varieties recently developed in the United States which have resistance of this general type are Sebago (36), Menominee (43), and Calrose (10).

Much higher resistance in certain of the wild species of *Solanum* has been recognized, but for a long time little emphasis was placed upon such species as breeding parents. One reason for this is that many have different chromosome numbers, and interspecific hybrids are difficult to handle. In recent years considerable attention has been given to *S. demissum* L. ($2n = 72$). Certain lines of this species are resistant, others susceptible, to *P. infestans*. Lehmann (25) studied the inheritance of this resistance and found it to be dominant and monogenic for each of two strains of the pathogen studied. In multiple hybrids with several wild species and

S. tuberosum, Black (6) suggested two major genes for resistance supplemented by several modifying genes. When highly resistant *demissum* parents are crossed with *tuberosum*, the F_1 is resistant. By backcrossing to *tuberosum* and eliminating susceptibles, desirable tuber types carrying the resistance of *demissum* can be built up. The resistance of *demissum* is very complete, necrotic spots appearing promptly following penetration, with the result that the fungus seldom survives and fruits (25). The first commercial potato variety carrying this type of resistance developed in the United States is Empire, introduced in 1945 by Reddick and Peterson (33). Since then, Reddick has introduced others with this type of resistance under the names Ashworth, Cheanago, Essex, Placid, and Virgil; and Akeley *et al.* have introduced Kennebec (1).

Variability in pathogenicity of *P. infestans* was first noted in 1919 by Giddings and Berg (20), who found in West Virginia, where tomato late blight is common, that the organism, as usually isolated from potato, was weakly pathogenic on tomato, while isolates from tomato were strongly pathogenic on both hosts. They considered that there were at least two physiologic races of the organism. In the 1930's potato breeders in Germany began to report that highly tolerant varieties failed to show much difference from susceptible varieties when tested in some localities. This led to a study of isolates, and it was found that some were more virulent on resistant varieties than were others. In the United States, at about the same time, Reddick and Mills (32) observed that the pathogen became progressively more virulent on resistant varieties in a given season. They demonstrated that when the wild type was passed through several generations on the foliage of a resistant variety (5 to 10 days being required for a generation), the virulence increased with each generation up to a certain level, at which point the resistance of the variety concerned appeared to be much less marked. This explained to some extent the experiences noted in Germany and offered an explanation for the decline in resistance of many varieties of former decades, beginning with Magnum Bonum cited above. One very important fact is that isolates with high virulence had no effect upon resistant *S. demissum* other than the necrotic reaction. Furthermore, new varieties derived from *demissum* appear to retain to a marked degree this high type of resistance, which, if it endures, will give them a decidedly superior advantage under optimum conditions for development of late blight.

Mills (28) has shown that the same phenomenon is encountered in the tomato strain. When an isolate from potato, weakly pathogenic on tomato, is passed through several generations on the latter host, it becomes highly virulent. It was shown later that the tomato strain is, at times, carried between seasons in potato tubers. The common observation that

tomato shows little or no damage when growing alongside potatoes severely affected by late blight was explained by Mills by the fact that the current strain of the pathogen has not had time to become acclimated to and virulent upon tomato. The tomato-late-blight epidemic of 1946 has not been fully explained, but it would appear that one or all of three possibilities entered into the situation: (*a*) continuously favorable weather began earlier than usual and provided greater opportunity for local increase of virulence of the pathogen; (*b*) carry-over of the tomato-virulent strain occurred on potato and was introduced early; (*c*) the tomato strain became established in southern-grown seedbeds and was distributed widely with transplants.

Control. Resistant varieties, when they meet the commercial requirement, are an important control measure for potato, but in tomato no suitable resistant varieties are available. Among potato varieties those recently introduced in which *demissum* resistance has been fixed offer promise of reasonably complete control. With other resistant varieties only partial control may be expected in severe disease epidemics.

Bordeaux mixture came into use rapidly after 1885 in Europe and America for control of late blight. For further details on the various fungicides mentioned below refer to Chap. 16. When the foliage is properly protected by adequate spraying, control is very efficient. When foliage is heavy and weather continuously rainy, difficulty in applying sprays is an important practical matter. Since dusters are lighter and more rapid, much experimentation has been given to dust substitutes. Copper-lime dust is effective, but it must be applied when dew is present. Insoluble coppers as sprays and dusts have not been so generally effective as Bordeaux mixture. The latter does cause some injury, and, this being due in part to increased transpiration, it becomes more of a factor in warmer, less humid areas. Much experimentation has been carried out with newer fungicides as substitutes for Bordeaux mixture. It is evident that no one material will be the most effective in every region or in every season in a given region. It becomes the more difficult, therefore, to give a general spray recommendation. In northern, more humid areas, such as New England, Pennsylvania, and the British Isles, the greater fungicidal value and tenacity of Bordeaux mixture has kept the latter in the forefront. In other regions, notably in the winter crop in Florida, dithane plus zinc sulfate and Parzate are often the most effective. Foliage protection and yield are not always correlated, owing to various factors influencing production, such as insect control and damage by other foliage pathogens, particularly that of early blight.

Bordeaux mixture is more toxic to tomato than to potato. Other fungicides have been sought more generally for the former on that account.

Here again some materials are effective for late blight but less so for early blight, blotch, *Septoria lycopersici* Speg., and anthracnose, *Colletotrichum phomoides* (Sacc.) Chester, which in many areas are more common than late blight. The insoluble-copper fungicides are less phytotoxic than Bordeaux mixture and are quite protective against late blight but they are not generally satisfactory for early blight. With some of the carbamates, *e.g.*, Zerlate, the opposite is the case. Therefore, an alternating schedule of an insoluble copper or Bordeaux mixture and Zerlate is recommended in some areas.

The forecasting of late-blight epidemics has been discussed under Disease Cycle. This type of service is important in fungicidal control in that it facilitates more timely applications and thus brings about savings in materials and more efficient protection. Cook (12) estimated a saving of 2 million dollars in fungicides in eastern Virginia in 1947 as a result of a forecasting service for late blight of potato and tomato.

When successful protection of potato foliage occurs, there often follows a serious problem owing to continuous production of inoculum up to harvest time and consequent contamination of tubers during lifting. This may offset to a considerable degree the benefits from control on the foliage. In the northern cool humid areas killing of foliage a few days before harvest is beneficial, and it has become a common practice to accomplish this by spraying with suitable herbicides, such as Sinox or Dow 66, or by using flame throwers. In England tar acids or sulfuric acid are used.

Under Disease Cycle the importance of timing harvest to minimize tuber contamination has been emphasized. This becomes an important control measure. Since bruising facilitates infection, avoidance of injuries at harvest is important. Sorting of visibly infected tubers before storage should be practiced. Where potatoes are stored in pits or clamps as in England, it is important to replace the straw covering after the sweating period in the autumn and before earthing up, and to gauge uncovering in the spring in relation to possible overheating (44).

Treatment immediately after lifting of tubers to be used for seed has given good control of tuber rot in England. Mercuric chloride, 1–1,000, for 90 min. or an organic mercurial was used (21).

The importance of sanitary measures in the disposal of tuber refuse from pits and storehouses to prevent such material from becoming a source of primary inoculum has been discussed under Disease Cycle.

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WHITE RUST OF CRUCIFERS

The white rusts are incited by several species of *Albugo*, which is the single genus making up the family Albuginaceae. These diseases, being common and conspicuous, were observed and studied by early mycologists including Persoon, Unger, Berkeley, Tulasne, and De Bary. The disease which affects many species of the Cruciferae and some species of the Capparidaceae is perhaps the most widespread. It occurs on many cruciferous weeds, probably most commonly on shepherd's-purse. Horse-

radish and radish are common economic hosts. Cabbage and cauliflower are affected in Europe but rarely in America.

The disease is relatively unimportant economically in proportion to its widespread occurrence. It is sometimes severe in northern states on radishes grown in greenhouses during winter months and on horseradish plantings. In radish, cabbage, and cauliflower seed crops the distortion of branches and flower parts may cause heavy losses.

Other economically important diseases incited by related species of *Albugo* are white rust of spinach, *A. occidentalis* G. W. Wilson (13, 14), white rust of sweet potato, *A. ipomoeae-panduranеae* (Schw.) Swing. or *A. minor* (Speg.) Cif. (3), and white rust of salsify, *A. tragopogonis* (DC.) S. F. Gray (14).

Symptoms. The effects upon the host plant result from two types of infection: local and systemic. In the case of local infection isolated pustules, or **sori** (singular, **sorus**), develop on leaves and stems. They are raised, white, shiny areas 1 to 2 mm. in diameter. They may arise in close proximity and merge to form larger patches. The host epidermis ruptures, but often not until some time after the pustule is fully formed. When this does occur, the pustule has a powdery consistency. When young stems and flowering parts are infected, the fungus becomes systemic in the tissue and stimulates hypertrophy and hyperplasia. This results in enlarged and variously distorted organs (Fig. 50). The distortion and abnormal development are particularly conspicuous on flower parts. Sepals become enlarged to several times normal size. Petals enlarge, and chlorophyll rather than the usual flower pigment is formed. Pistils and anthers are likewise distorted, and normal seed development is prevented. In a given flower some parts may be normal, others greatly distorted. Pustules may form on hypertrophied organs, but they may be relatively rare on such tissue.

The Causal Organism. *Albugo candida* (Lév.) Kunze, 1891.

Synonymy:

Aecidium candidum Pers., 1791.
Uredo candida Pers., 1801.
Uredo cheiranthi Pers., 1801.
Cystopus candidus Lév., 1848.

The members of the Albuginaceae are distinguished from those of related families by the formation of the asexual sporangia (or conidia) in chains. The mycelium is intercellular in the host tissue except for knob-shaped intracellular haustoria. When a pustule, or sorus, forms, numerous short sporangiophores arise from the mycelium in a closely compacted palisade layer beneath the epidermis and at a right angle with it (Fig. 51).

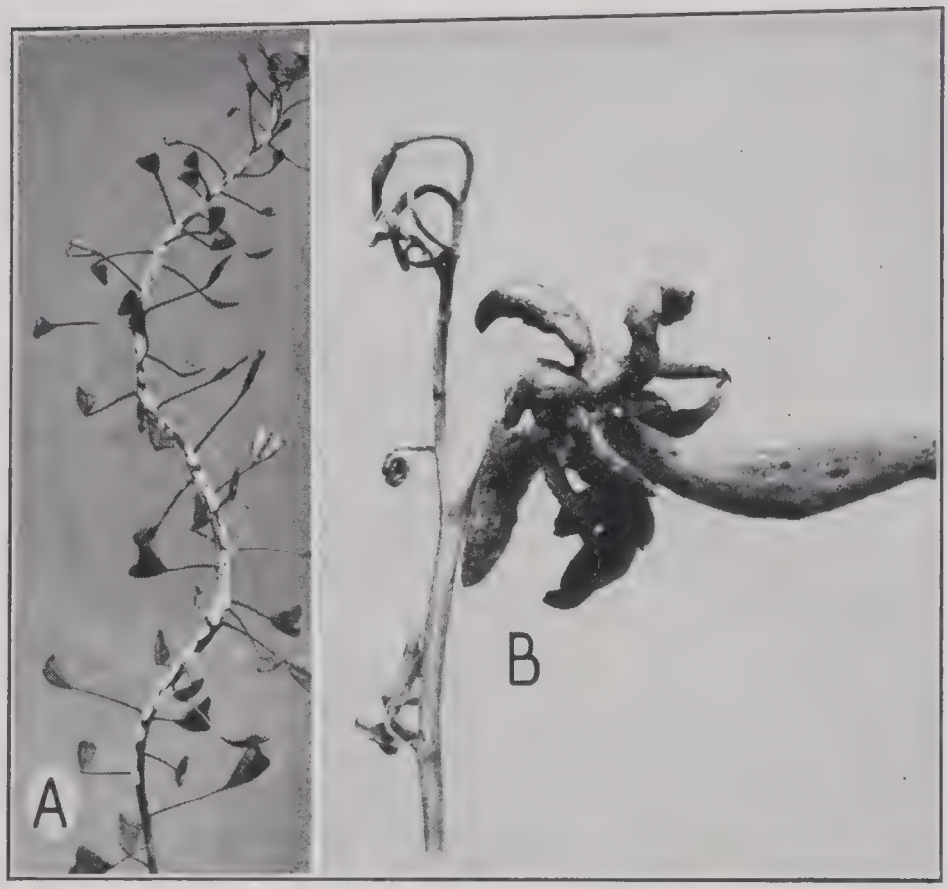

FIG. 50. White rust of crucifers. *A*, local lesions on shepherd's-purse; *B*, systemic invasion of radish flower, causing hypertrophy of sepals, petals, and ovary.

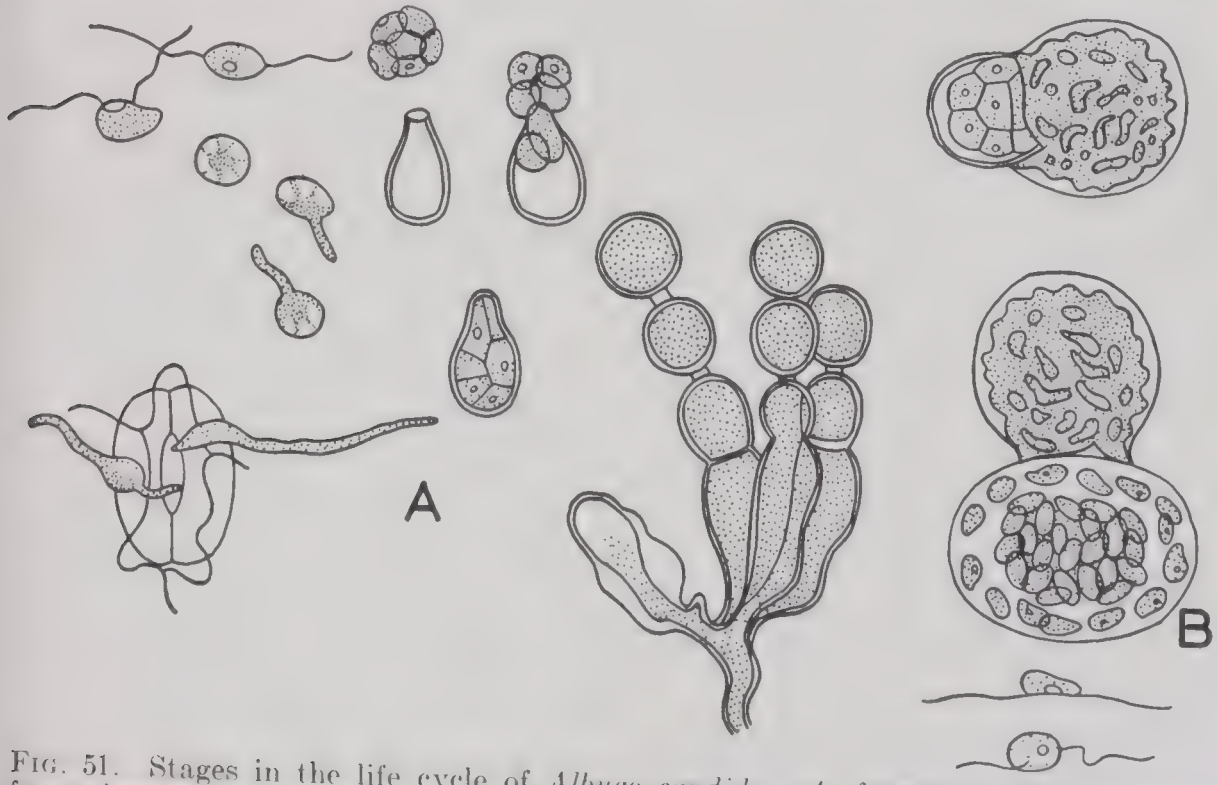

FIG. 51. Stages in the life cycle of *Albugo candida*. *A*, formation of sporangia, formation of zoospores, and stomatal penetration by germ tubes from encysted zoospores; *B*, germination of the oospore by formation of a sporangium and zoospores. (*After De Bary.*)

The first sporangium is formed at the tip of the sporangiophore, and others are formed successively beneath; pads of gelatinous material formed between successive spores function as disjunctors. The chains of spores may be fully formed, and in some species germination of sporangia may take place, apparently, before the sorus ruptures. The sporangia of *A. candida* are hyaline, nearly spherical, and 14 to 16 by 16 to 20 μ in size. They germinate by means of a germ tube or by the formation of zoospores. The former method appears to be exceedingly rare. Formation of zoospores was first recorded by Prévost in 1807. Tulasne in 1854 and Hoffman in 1859 were unable to confirm Prévost and described germination by germ tubes. De Bary in 1860 described for the first time the details of zoospore

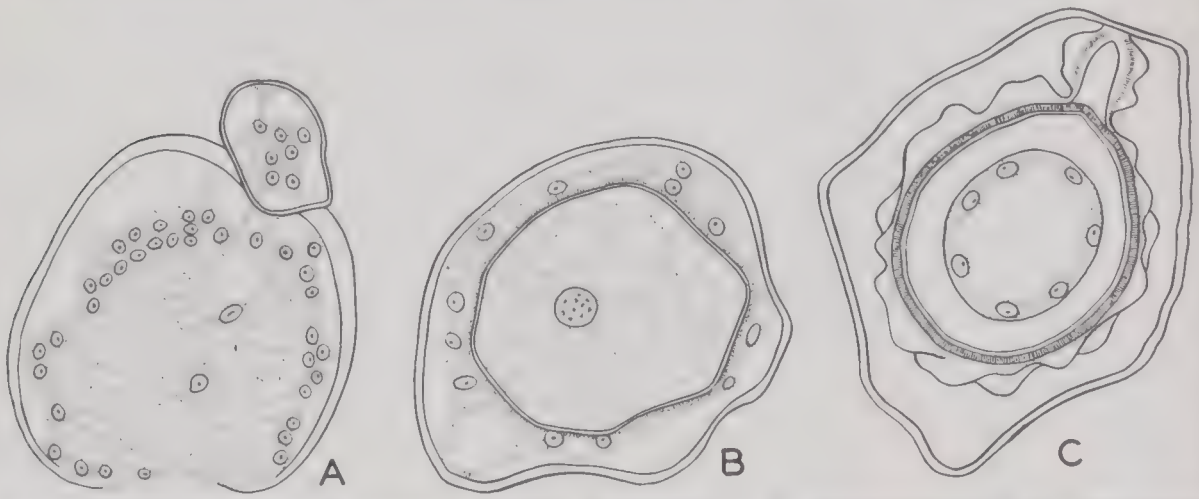

FIG. 52. Formation of oospore of *Albugo candida*. *A*, oogonium and antheridium with fertilization tube. The egg nucleus is in the center; the apical wall of the tube is becoming very thin, and the antheridial nucleus is about to enter the oogonium. *B*, the oospore wall has begun to form; the fusion nucleus lies at the center. *C*, the ripe oospore in cross section, showing 7 of the 32 nuclei in the parietal layer of the protoplasm. The remains of the fertilization tube are shown surrounded by the exospore. Immediately within the latter is the endospore. (*After Wager.*)

formation (7). As the spores absorb water, they swell; vacuoles in the granular protoplasm form and then disappear, and finally five to eight polyhedral portions of the protoplasm are delineated by fine lines. In the meantime, an obtuse papilla is formed at one side of the spore, which now swells and opens while the zoospores, still immobile, emerge usually one by one, final cleavage sometimes following complete emergence of the contents of the spore. The flagella soon become apparent by an oscillatory motion of the entire spore mass. The slightly concave-convex zoospore contains a disk-like vacuole on one side, near which are attached two cilia, one short and one long, by which the spore soon detaches itself from the mass and swims away if liquid is present.

The oogonia and antheridia are formed from the mycelium in the intercellular spaces of the host, particularly in systemically invaded tissue

(12). The oogonium is globose, terminal or intercalary, and contains about 100 nuclei. The contents of the oogonium are clearly defined into a peripheral zone of periplasm and a single central oosphere. The antheridium is clavate, contains 6 to 12 nuclei, and is applied to the side of the oogonium. At the point of contact the wall becomes very thin, and a papilla of the oogonium protrudes into the antheridium and soon disappears (Fig. 52). The fertilization tube from the antheridium passes through the thin spot in the oogonial wall to the oosphere. The nuclei in the oosphere and in the antheridium undergo two mitotic divisions; those nuclei in the periplasm disorganize. A granular body known as the coenocentrum appears in the oosphere, and a single nucleus is attracted to a point near it. The fertilization tube penetrates to the coenocentrum, ruptures, and discharges a single nucleus, which fuses with the oogonial nucleus. The fertilization tube collapses, and the coenocentrum disappears. A wall is now formed around the oosphere to constitute the oospore. The wall is eventually thick and tuberculate. The fusion nucleus later divides, and division continues until about 30 nuclei are formed. Tsang (11) noted about 24 chromosomes in the anaphase of the first division of the fusion nucleus and 12 in the second anaphase. The reduction division appears to take place, then, in the first division of the fusion nucleus. The oospore acts as a resting spore. De Bary described germination by formation of a vesicle in which zoospores are formed. No other investigators have reported oospore germination.

Disease Cycle. All species of *Albugo* are obligate parasites. Overwintering is probably chiefly by means of oospores. In perennial hosts, such as horseradish, the mycelium persists in the crowns and occasionally in lateral roots (5). Secondary spread is by means of sporangia, which are readily carried by air currents. Eberhardt (2) showed that sporangia germinate most readily at the time the pustules break open. Melhus (7) was the first to show that chilling of the spores is essential to bring about production of zoospores. After chilling, germination occurs over a range of 1 to 20°C. Napper (8) found that reduction of the water content of the spores to about 30 per cent was essential for germination, and the more rapid the rate of dehydration, the prompter the germination. Moisture on the host surface is essential for germination and infection.

Motile zoospores swim for a short time, become quiescent, and produce germ tubes. Invasion takes place through stomata. The germ tubes enter stomata of resistant hosts as readily as those of susceptible hosts. In the former, growth of the pathogen ceases in the substomatal chamber; in the congenial or susceptible host the mycelium advances intercellularly with the production of haustoria.

Many workers since Eberhardt have observed that in *A. candida* and in some other species, physiologic races occur. The extent of specialization

and the number of races have never been fully investigated. Togashi and Shibasaki (10) found the sporangia from races on *Brassica* and *Raphanus* to be larger than those from *Capsella* and *Arabis* and suggested the subspecies *macrospora* and *microspora*. The race on radish is restricted rather closely to the genus *Raphanus*; that on cabbage, cauliflower, and other members of *Brassica oleracea* L. to that group of hosts; and the form on horseradish also has a narrowly limited host range (5).

Control. The disease is of such limited economic importance that no effective control measures, even for radish and horseradish, have been worked out.

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THE DOWNY MILDEWS

The downy-mildew fungi make up the family Peronosporaceae. They are important plant pathogens and are all obligate parasites. Most of them produce oospores within the host tissue. Each oospore is formed in a single-celled oogonium fertilized by a single antheridium, very similar to that described for the genus *Albugo*. In asexual reproduction the mycelium produces branched sporangiophores of determinate growth, which

emerge through the stomata of the host epidermis. Sporangia are borne singly at the tips of the ultimate branches and, when mature, are readily dislodged. The diseases are characterized by the downy growth on the host lesions, consisting of sporangiophores and sporangia. The genera of the Peronosporaceae are distinguished from one another chiefly by the form of the sporangiophores. The characteristics of the chief genera are given below:

Sclerospora. Sporangiophore stout with heavy branches clustered at the apex. Sporangia germinate by germ tubes or zoospores. Example: *Sclerospora graminicola* (Sacc.) Schroet., incitant of downy mildew of grasses.

Plasmopara. Sporangiophore monopodial to subdichotomous, with branches arising more or less at right angles, tips obtuse. Example: *Plasmopara viticola* (Berk. & Curt.) Berl. & DeT., incitant of grape mildew.

Peronospora. Sporangiophore dichotomously branched, tips acute, sporangia not papillate or only slightly so. Example: *Peronospora destructor* (Berk.) Casp., incitant of downy mildew of onion.

Pseudoperonospora. Sporangiophore subdichotomously branched, branches arising at acute angles, with subacute tips, sporangia papillate. Example: *Pseudoperonospora cubensis* (Berk. & Curt.) Rostow., incitant of downy mildew of cucurbits.

Bremia. Sporangiophore dichotomously branched, with disk-like tips from the border of which sterigmata arise bearing sporangia with apical papillae. Sporangia germinate by zoospores or by germ tubes. Example: *Bremia lactucae* Regel, incitant of downy mildew of lettuce.

DOWNY MILDEW OF GRASSES

The downy mildews incited by members of the genus *Sclerospora* are most important in tropical and subtropical areas, particularly in the Philippine Islands, Java, and India. The hosts are chiefly in the grass family and include such important crop plants as maize, sugar cane, sorghum, and millet. The species *Sclerospora graminicola* considered in detail here is the most widespread, occurring in both tropical and temperate zones of America and Eurasia. The disease was reported in Europe by Schroeter (8) in 1879. It was described in America in 1884 by Farlow (6) on green-foxtail grass, *Setaria viridis* (L.) Beauv., collected near La Crosse, Wisconsin. It was first noted on maize in Wisconsin in 1921 (6). The host range includes several species of *Setaria* and some other species within the tribes Paniceae and Tripsoceae of the family Gramineae, including teosinte and maize. It is of economic importance on Italian millet in Japan and Korea.

Symptoms. Plants that are systemically infected show chlorotic streaks in some of the leaves, while others remain normal. The tissue later turns brown and tends to become shattered. Stunting of growth is

a prominent feature. The systemically infected inflorescence, as well as the leaves in proximity to it, may show various degrees of malformation and overgrowth. The appearance of mildew on the lesions may be inconspicuous, since it tends to develop chiefly during the night when dew is present and to wither promptly during bright clear daylight. Some infected leaves may bear the mildew, which consists of sporangiophores and sporangia, while in other infected leaves only oospores are produced within the tissue. The latter are the more likely to shatter with age. Weston and Weber (14) described local lesions on everglade millet which consist of somewhat sunken spots, variable in size and in shape. In general they are elongate and rather sharply bounded laterally by the leaf veins. The surface of the lesion is irregularly roughened, and the color is buff to dull reddish to very dark brown. Production of mildew takes place chiefly from the undersurface of the spots. They are otherwise easily confused with other fungus or bacterial leaf spots.

The Causal Organism. *Sclerospora graminicola* (Sacc.) Schroet., 1879.

Synonymy:

Protomyces graminicola Sacc.
Peronospora graminicola Sacc.

Although this is the type species of the genus, it is quite distinct from other species in that the sporangia germinate principally by zoospores. The sporangiophores arise from intercellular mycelium in the mesophyll tissue of the leaf, emerging in clusters through the stomata. Small bulbous haustoria occur. The sporangiophore consists of a single stalk, which increases in diameter as it expands to as much as 30 μ at the base of the short sporiferous branches which comprise the head (Fig. 53). The branch which coincides most closely with the main axis tends to be somewhat longer than any of the others. After two or three further dichotomous branchings, the tenpin-shaped, short sterigmata arise. From the extreme tip of each sterigma a sporangium develops until it is cut off at maturity by a septum. There is some evidence that forcible discharge of sporangia occurs. Sporangia are mostly broadly ellipsoid to broadly cylindric. While a wide range in size may be found in a large population, most of the sporangia are within the measurements of 12 to 16 by 16 to 22 μ. The sporangium is thin-walled and hyaline with a characteristic papilla at the distal end. The latter is a ready means of distinguishing this species from others in the genus.

The sporangia germinate promptly under favorable conditions by the production of one to eight, usually three to four, biciliate zoospores, which emerge through the papilla. The encysted zoospores are 9 to 12 μ in

diameter. The details of oospore formation are described by McDonough (4). In the early stages the development of the oogonium is similar to that of *Albugo candida*. It is usually terminal, occasionally intercalary. The protoplasm including about 50 nuclei enters the oogonium from the mycelium, and after the expansion of the oogonium it is cut off by a septum.

FIG. 53. Three stages in the formation of the sporangia of *Sclerospora graminicola*. (*After Weston.*)

The nuclei are at first shriveled in appearance, but many enlarge and, as they advance to the metaphase, become oriented in the region which is to become the boundary of ooplasm and periplasm. The nuclei pass through two mitotic divisions. The first division is simultaneous in all nuclei, but in the second division it is not necessarily so. A coenocentrum develops, near which a single nucleus is found, which, in turn, divides; one daughter nucleus later serves as the female nucleus. The antheridium contains three to four nuclei, which divide twice, mitotically, but slightly

in advance of division in the oogonium. There is no receptive papilla in the oogonium as in *A. candida*, the fertilization tube penetrating the oogonial wall and passing rapidly to the oosphere, where it discharges one nucleus and promptly disintegrates. The coenocentrum disappears, and the single oogonial nucleus and the antheridial nucleus remain apart for some time. Differentiation of the oospore wall follows by secretion from the ooplasm. A thin outer layer makes up the exospore, and a thicker layer comprises the endospore, while the old oogonial wall collapses to touch the new oospore wall in some places. Most of the oospores fall within the limits of 30 to 36 μ in diameter. The binucleate condition may persist until after the resting period. A central body develops in the mature oospore which is not unlike that seen in other phycomycetous oospores. McDonough (5) believes this to consist chiefly of carbohydrate or of a carbohydrate-protein complex rather than of fat. It begins to show signs of digestion previous to germination, and the oily content of the oospore increases simultaneously. The wall becomes thinner at this time, reducing from about 4 to about 1 μ in thickness. Two germ tubes were reported by Hiura (3), but McDonough never found more than one. It arises invariably through the thin spot in the wall marking the point of entrance of the fertilization tube. After fusion of the oogonial and antheridial nuclei and during the early stage of germination, the nucleolus of the fusion nucleus enlarges. In mitotic divisions of nuclei there are 14 to 16 chromosomes while in the prophase of the fusion nuclei McDonough (4) found at least 28 chromosomes. At meiosis 14 moved to each pole, and in the next two subsequent divisions there were 14 at each pole. Reduction therefore takes place in the first division after fusion. About 32 nuclei are seen in the oospore before the germ-tube formation is complete, and they pass into the latter with all the cytoplasm of the oospore.

Physiologic races have been reported by Tasugi (10) in Japan and by Uppal and Desai (12) in India.

Disease Cycle. Melhus *et al.* (6) showed that infection upon young seedlings took place from oospores in the soil from the time the testa was broken until the plumule had emerged above ground. The oospores appear to be the chief means of overwintering in northern temperate regions. Tasugi (10) reported seed transmission of oospores. Hiura (3) reported infection through roots, coleoptiles, and rhizomes, the susceptibility of each organ decreasing rapidly with age. He found dry soil to be more favorable than moist soil and the minimum, optimum, and maximum soil temperatures to be 11°, 20°, and 34°C., respectively. Pu and Szu (7) found 24 to 32° during the first 2 days after sowing to be the most conducive to heavy infection.

Secondary inoculum consists of sporangia. Weston (13) found that production of sporangia took place only at night and when dew was present, and this was in accord with the situation with certain tropical species of *Sclerospora*. Melhus *et al.* (6) and Tasugi (9), however, found sporulation to occur in daylight if high humidity was provided. Sporangiophores mature in about 2 hr., and sporangia mature in another 1 to 2 hr. The minimum, optimum, and maximum temperatures for formation of sporangiophores and sporangia are about 5°, 18 to 20°, and 35°, respectively. Sporangia germinate over a range of 5 to 33°, the optimum being about 17°. At the optimum, germination occurs in about 1 hr. At the low and high ends of the range, and with old conidia at any temperature, germination is only occasionally indirect and mostly by means of a germ tube.

Control. Since the disease has not been of great importance on economic hosts in the United States, there has been little or no attention given to the development of control measures. In China and Korea the severity of the disease on italian millet, *Setaria italica* (L.) Beauv., has prompted a study of the possibility of control through disease resistance. Yu (15) tested several varieties and found Tsinan-Nanking No. 2 highly resistant to mildew as well as to kernel smut, *Ustilago crameri* Koern. It is recommended for use in regions of North China.

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DOWNY MILDEW OF GRAPE

The downy mildew of grape is endemic in North America, where it was first recorded by Schweinitz in 1837. It is most common in the eastern states. While the pathogen has been recorded in California, mildew is of no importance there. The disease was first noted in Europe in 1878, when it was discovered by Millardet near Bordeaux, France, in a nursery of seedlings which had been imported from the United States. In western Europe it encountered a favorable environment, and the varieties of grape under culture were, as a rule, much more susceptible than the varieties in the eastern United States. Downy mildew rapidly became a serious disease of grape in Europe, and had not Bordeaux mixture been developed between 1882 and 1885 by Millardet, losses would probably have been tremendous. In spite of fairly satisfactory control measures the disease remains a very important one in many intensive grape-growing regions.

The fungus occurs on many species of *Vitis*, on the five-leaved ivy (*Parthenocissus quinquefolia* Planch.), and on English ivy (*P. tricuspidata* Planch.).

Symptoms. The first sign of the disease on the leaf is a small greenish-yellow oily-appearing spot on the upper side which enlarges to $^1_2$ cm. or more in diameter. The tissue in the spot is traversed by reddish lines, and the whole area later becomes brown and brittle. On the lower side of the leaf, the downy mildew consisting of the sporangiophores and sporangia of the pathogen appears soon after the spot develops (Fig. 54). In moist weather it persists; in extremely dry weather it may disappear. Leaves with many active spots may drop prematurely.

Young shoots and tendrils may be affected, and the fungus, becoming systemic, stunts growth and fruits heavily on the surface. The young berries show brownish spots and later become covered with downy growth. On older fruits brownish patches appear, and the berry may harden. Later the skin shrivels, and the color becomes grayish blue or dark brown. The

fruit shells extensively if the attack is severe. The damage to growing canes and to fruit may be very heavy. Storage decay is not induced by the mildew pathogen.

The Causal Organism. *Plasmopara viticola* (Berk. & Curt.) Berl. & DeT., 1898.

FIG. 54. Downy mildew of grape.

SYNONYMY:

Botrytis cana Lk., according to Schweinitz, 1837, not *B. cana* Kunze & Schm.
Botrytis viticola Berk. & Curt., 1855.
Peronospora viticola (Berk. & Curt.) Casp. (1855?), 1863.

Sporangia are borne terminally on sporangiophores which branch monopodially along the main trunk and dichotomously at the extremities (Fig. 55). The sporangia are hyaline, ovate, and attached to the sporangiophore at the small end, where the wall is thickest. At the opposite end is a papilla, in which the wall is thinnest. The protoplasm is finely granular. After about an hour in water, light hyaline spots appear while the rest of the protoplasm becomes denser, and finally dark lines appear which delineate the cell contents around each spot. The swarm spores are formed promptly, and the entire mass is extruded through the papilla, the thin

wall of which is dissolved or ruptured. The flagella of the spores cause a jerking and wrenching movement until the zoospores become separated and swim away. Up to 17 biflagellate spores are produced in a sporangium. The zoospores (6 to 7 by 7.5 to 9 μ) are plano-convex with a ridge along the flat side, near which is a light hyaline spot, the nucleus. The flagella are of unequal length (27 to 33 μ) and arise on either side of the nucleus. After about 30 min. activity the zoospores come to rest, absorb the flagella, and in about a quarter hour produce a germ tube.

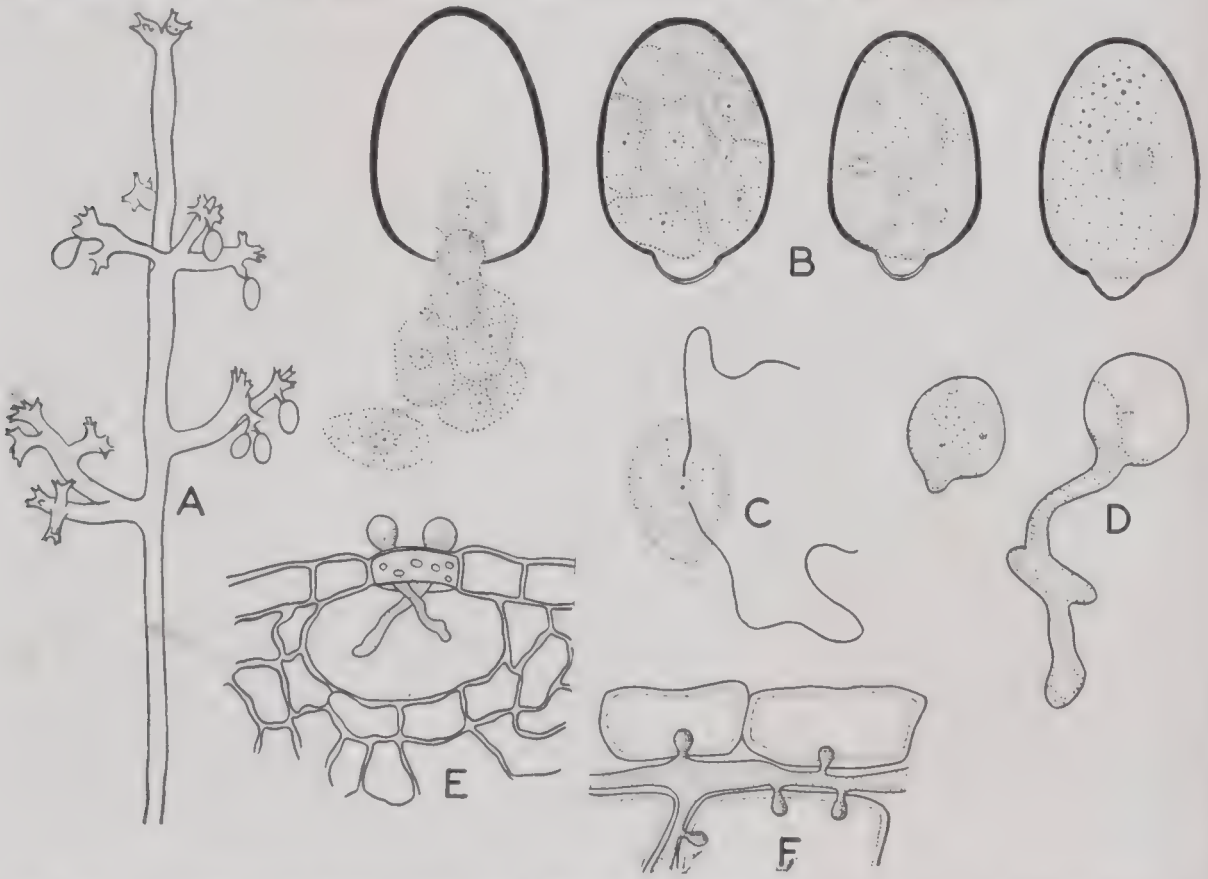

FIG. 55. *Plasmopara viticola*. *A*, sporangiophore; *B*, formation of zoospores; *C*, zoospore; *D*, germination of encysted zoospore; *E*, stomatal penetration by latter; *F*, intercellular mycelium and haustoria. (*A after Farlow; B–F after Gregory.*)

The fungus enters the stomata, and the coenocytic mycelium is intercellular except for the globose haustoria, which form abundantly. The oogonia are formed terminally, are spherical and about 30 μ in diameter. The clavate antheridium (40 to 50 by 20 to 25 μ) becomes attached to the side of the oogonium, and a fertilization tube is formed. A single nucleus from the antheridium enters and fuses with one of the nuclei in the oogonium. The fusion nuclei are about $2\frac{1}{2}$ times the size of other nuclei. The mature oospore is uninucleate. Bose (5) states that the reduction division of the nucleus to form the haploid phase takes place in the third or fourth division of nuclei in the oospore.

The oospore consists of a thick smooth endospore wall overlaid by a thinner rough exospore wall, upon which the oogonial wall has collapsed.

The oospores are to be found in infected canes, where they germinate in the spring months by production of a hypha which protrudes from a small crack in the oospore wall. At the tip of the hypha is produced an ovate sporangium (27 by 31 to 47 μ) not readily distinguished from the largest summer sporangia. Occasionally two hyphae may arise from an oospore. The sporangia apparently give rise to zoospores.

Disease Cycle. The oospores are the chief means of overwintering. Perennial mycelium in infected shoots has been reported in California (4). The oospores germinate in early spring, the time required for the process becoming less as the season advances up to midsummer, when little further germination is observed. Some spores remain viable until the second season. After primary infection has occurred, secondary spread is by means of sporangia, which are readily detached and disseminated by air currents. Yarwood (16) has shown that, humidity and temperature being favorable, sporulation occurs at night and that it is dependent upon physiologic conditions provided by the diurnal alternation of light and darkness.

The zoospores appear to be attracted to the stomata of leaves of the grape as well as of other plants which do not become hosts. Penetration of stomata of grape and nonhosts occurs, but in the latter no further development beyond the substomatal chamber takes place. In the susceptible host the mycelium invades the intercellular spaces, ramifying through the tissue and deriving its nutrients in a large measure through haustoria which penetrate the cell lumen.

Practically all infection of the leaves takes place through the lower side. In fact, Gregory (9), in carefully controlled experiments, secured no infection when inoculum was applied to the upper side as compared with an effectiveness of more than 50 per cent for the inoculations on the lower side.

The optimum temperature for germination of oospores is 20 to 25°C. Sporangia germinate in 1 to 2 hr. at the same range. The incubation period for production of sporangia after infection is 5 to 18 days depending upon the temperature and humidity. According to Müller and Sleumer (12) the optimum temperature for the development of an epidemic is 18 to 24°, the minimum 12 to 13°, and the maximum 30°. In the Baden area of Germany they developed a scheme of forecasting mildew epidemics by close study of prevailing humidity and temperature. The formula developed in Baden has not always been found to be applicable in other regions, indicating that different combinations of environal factors may be favorable for disease development.

Varietal Resistance. Generally speaking, European grape varieties (*Vitis vinifera* L.) are more susceptible to the pathogen than American stocks. Although a considerable amount of breeding for the development

of resistant varieties has been carried out in Europe since 1890, complete immunity has not been attained, but various degrees of resistance occur. However, none of the varieties which are satisfactory horticulturally in the vine-growing regions of Europe is sufficiently resistant, under optimum conditions for the disease, to preclude the need of application of fungicides. Previous to World War II an intensive breeding program was under way at the Muncheberg Plant Breeding Station in Germany. The progress up to 1938 is described by Scherz (13). By crossing of European with American grapes and continuous backcrossing to the European parent with elimination of all susceptible seedlings in each generation, distinct progress in transferring resistance to the European type was attained. By extensive screening of *V. vinifera* seedlings a gene for resistance within this species was also found. In America the natural resistance of varieties used in eastern states and the unfavorable environment for mildew where susceptible European varieties are used in western states have minimized the need for a breeding program for mildew resistance. The relative resistance of American and European varieties when tested in this country is summarized by Demaree *et al.* (6).

Control. The historical aspects of grape-mildew control and the discovery of Bordeaux mixture are discussed in Chaps. 15 and 16, respectively. In Europe the use of a protectant fungicide is a standard procedure. Bordeaux mixture is still the most reliable material when conditions are likely to be optimum for the disease. Insoluble coppers, while satisfactory under some circumstances, are not generally so reliable. When Bordeaux mixture is applied thoroughly and at proper intervals, a high degree of control may be attained.

The time of application varies with prevailing environment and the relative susceptibility of the variety concerned. When a variety is markedly resistant, spraying may be delayed longer than with a very susceptible sort. When the environment is suboptimum, the period between applications may be increased. The best results in European grape-growing regions appear to result where an advisory service is maintained which is based upon a close watch of weather conditions. Such a successful service in the Baden area of Germany is described by Müller and Sleumer (12) and in the Moselle area by Zillig (17).

In eastern United States the grape spraying schedule is based on control of black rot, *Guignardia bidwellii* (Ell.) Viala & Ravaz, and insects as well as of mildew. The former disease is often the most important. The general program for eastern United States, subject to local modification, is given by Demaree and Runner (7) as follows:

1. Spray with 4-4-50 Bordeaux mixture when shoots are 7 to 8 in. long.

2. Spray with the same, plus 1 lb. of calcium arsenate and $^1/_2$ pint of fish oil or linseed oil per 50 gal., 1 week after blooming.
3. Repeat (2) as the blossoms fall.
4. Two weeks after (3) spray with Bordeaux plus $^3/_8$ pint nicotine sulfate and oil as in (2) per 50 gal.
5. Repeat (4) when fruit are half grown. In New York State, when black rot is troublesome, Fermate is more satisfactory than Bordeaux mixture in the early sprays, but since the former is not so effective against mildew, the latter is retained for the later sprays.

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DOWNY MILDEW OF ONION

In 1841, Berkeley made the first record of this disease on a species of *Allium*, presumably on the onion, *A. cepa* L., in England. It was first reported in America in 1884 by Trelease, who found it in Wisconsin. It has been recorded since from many parts of the world. Where relatively cool moist weather prevails, it is often very destructive. In the British Isles and in other regions bordering the North Sea it is a major disease. In the United States it occurs most frequently and most severely in New England, New York, Louisiana, California, and Oregon. It is spasmodic in many other onion-growing regions on the bulb crop, the greens crop, and the seed crop.

Besides onion a number of other species of *Allium* are recorded as hosts, including Welsh onion (*A. fistulosum* L.), shallot (*A. ascalonicum* L.), leek (*A. porrum* L.), garlic (*A. sativum* L.), and chive (*A. schoenoprasum* L.).

Symptoms. Depending upon the means of overwintering, the disease appears on leaves growing from systemically infected plants or bulbs or as local lesions on leaves or stems, presumably from infection from overwintered oospores. Systemically infected plants are dwarfed, and the leaves tend to be distorted and pale green. In humid weather the fungus fruits over the surface of the leaves and seed stems, producing the violet downy mildew characteristic of the disease (Fig. 56). In dry climate or in a dry greenhouse white spots appear on systemically infected leaves.

Local lesions, resulting from secondary infection, are oval to cylindric in shape, variable in size, and often paler than the normal yellow green of the leaf or stem. They may consist of alternating chlorotic and green layers of tissue. In humid atmosphere the fungus fruits on the surface. In drier atmosphere the lesion may become necrotic in the center without fructification. When leaves are infected between the center and the tip, the leaf droops at the point of the lesion, and the tip dies. The plant produces new leaves as growth continues. The severity of the disease depends on the environment. Advance may be checked for a time in dry weather and then be renewed with the return of favorable conditions. Plants are seldom killed, but bulb growth is reduced, and the bulb tissue is inclined to be spongy and of poor keeping quality. When expanding seed stems are infected, uneven stunted growth follows. When growth is checked by infection on one side of the stem, the latter bends in the direction of the lesion. As the seed umbel becomes heavier, weakened stems break over, and either seed maturation is prevented or mature seed is shriveled.

A secondary organism (*Stemphylium botryosum* Wallr.) commonly de-

velops in onion-mildew lesions as a saprophyte or a very mild facultative parasite. The fructification of this organism consists of dark-colored spores produced in abundance on the surface of the lesion. The conspicuous appearance of this black mold is commonly such as to obscure the sporangiophores and sporangia which make up the mildew of the downy-mildew disease.

The Causal Organism. *Peronospora destructor* (Berk.) Casp., 1860.

SYNONYMY:

Botrytis destructor Berk., 1841.
Botrytis parasitica, Schleiden, 1846.
Peronospora schleideni Unger, 1847.
Peronospora schleideniana (Unger) DBy., 1863.
Peronospora schleideniana (Unger) f. *cepae*, Thuem, 1879.
Peronospora destructor Berk., 1943.

FIG. 56. Downy mildew of onion seed. stalks.

There are several points of view as to the correct binomial for the pathogen. The nomenclature of the fungus is discussed by Cook (1) and by Yarwood (11). The name recommended by the former is followed here.

The sporangiophores are nonseptate, variable in length (122 to 820 μ), and swollen at the base to a diameter of 7 to 18 μ. Branching is dichotomous, with subacute to acute sterigmata bearing pyriform to fusiform sporangia (18 to 29 by 40 to 72 μ) attached by the pointed ends (Fig. 57). The sporangia are thin-walled, subhyaline, and slightly papillate at the distal end. The sporangia do not form zoospores but germinate by means of one or two germ tubes. The nonseptate mycelium is intercellular with filamentous haustoria. The oogonia, formed intercellularly, are 43 to 54 μ in diameter. Oospores are 40 to 44 μ in diameter. They germinate by means of a germ tube.

Although the question of physiologic specialization of the fungus has received little study, it is evident that races exist, since some hosts have been reported free from the disease where other hosts were severely affected.

Disease Cycle. The fungus overwinters as systemic mycelium in bulbs and in infected overwintering plants in mild-winter areas and as oospores in the soil. Since infection of flower parts has been found, and since oospores have been reported on the surface of seeds, some workers have regarded seed transmission as important. Yarwood (11) however, found no evidence of such transmission of the pathogen.

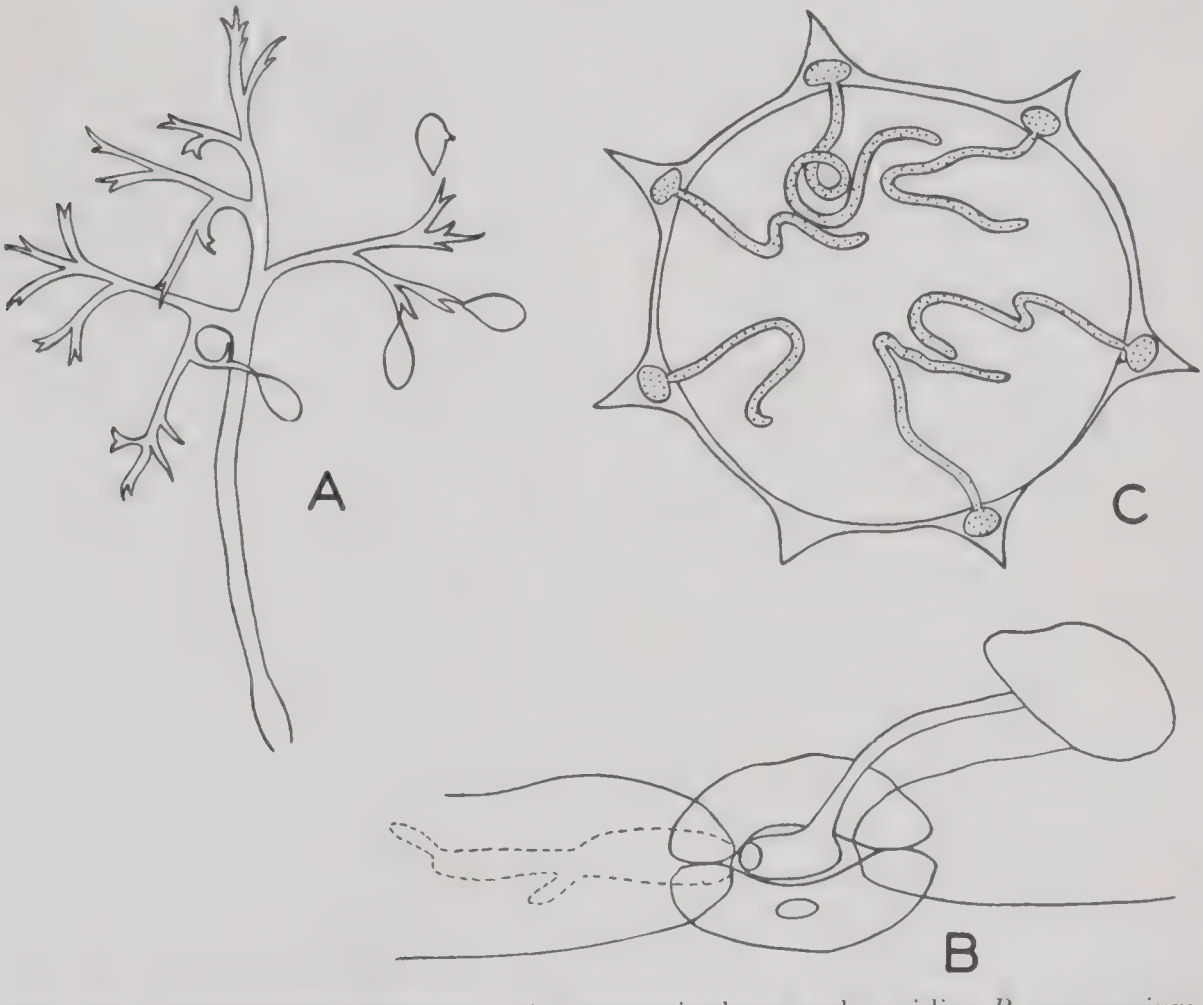

FIG. 57. *Peronospora destructor*. *A*, sporangiophore and conidia; *B*, sporangium germinating by a germ tube, the latter penetrating a stoma; *C*, intercellular mycelium in cross section and haustoria. (*After Yarwood.*)

The germ tube usually forms an appressorium over the stomatal opening, penetrates through the stoma, and forms a substomatal vesicle from which the intercellular mycelium arises and sends filamentous haustoria into the host cells.

Sporangia are produced in high humidity over a range of 4 to 25°C., with an optimum at about 13°. Yarwood (11) found sporangia to develop during the night, maturing early in the morning and being disseminated during the day. Both relative humidity and alternation of light and darkness influence sporulation. The sporangia remain viable, when attached,

for about 3 days, or even longer in the dark, but for a shorter period when detached. Air dissemination is the chief means of spread. Germination occurs in water from 1 to 28° with little difference in percentage of spores germinating from 7 to 16°. Relatively cool and rather humid weather is required to build up an epidemic. Rain is not necessary if heavy dews occur repeatedly.

Control. Although many control experiments with inorganic and organic fungicides show a reduction in the amount of disease, control by these means has seldom been applied successfully. Factors which seem to have reduced the effectiveness of this type of control measure are the spasmodic nature of the disease, the difficulty in getting fungicides to stick on the glossy surface of onion leaves, and the rapid exposure of new leaf tissue during the growing period.

Avoidance of poor air drainage has a beneficial effect. Eradication of perennial onions as a source of inoculum is important in some sections. Seed growing has shifted to some extent to semiarid irrigated areas to reduce the mildew hazard.

While differences between varieties in their susceptibility to mildew has been observed by numerous workers, little use of resistance as a control measure has been made until recently. Calred is a variety introduced by the California Experiment Station and the U.S. Department of Agriculture in 1947. It is derived from a cross between a resistant male-sterile line of Italian Red and Lord Howe Island. The seedstalks are highly resistant and the leaves moderately so. More resistant varieties may be expected to be developed from the same parentage.

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DOWNY MILDEW OF CUCURBITS

This disease was first described in 1868 on the basis of specimens of a cucurbitaceous plant collected in Cuba and sent to Berkeley in England. It was not recorded again until 1889, when Halsted described it on cucumbers growing in a greenhouse in New Jersey and when Farlow described it on specimens of cucumber from Japan. During this and the next year it was reported at various points from Massachusetts to Florida and Texas. In the United States it still occurs as a major disease chiefly along the Atlantic seaboard.

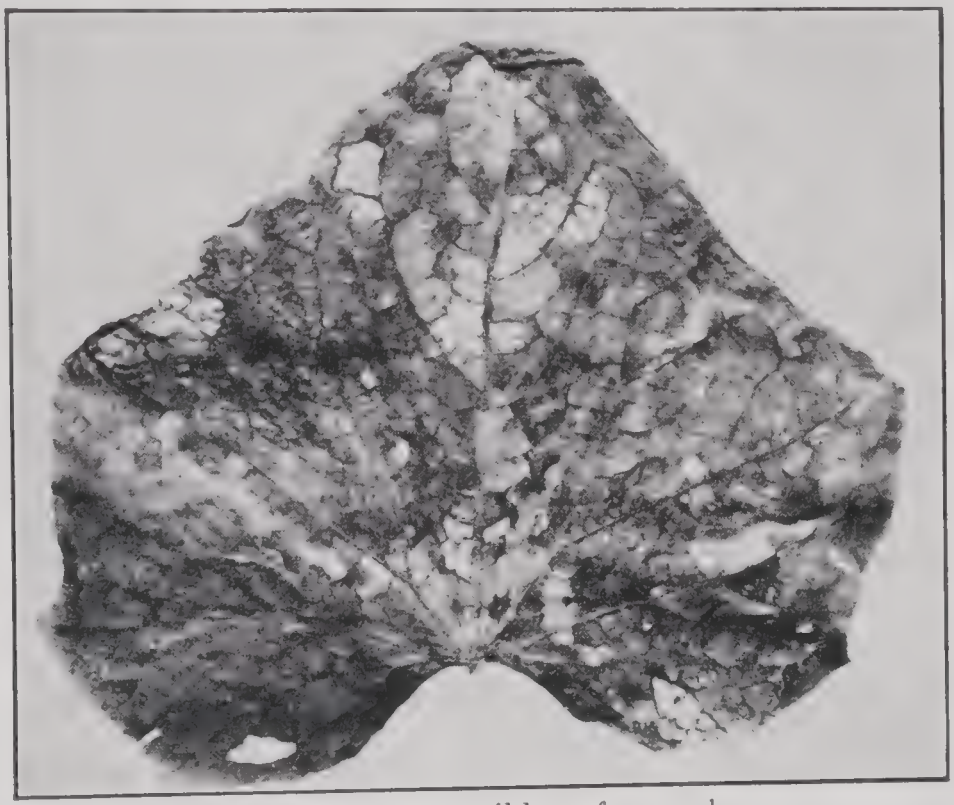

Fig. 58. Downy mildew of cucumber.

Cucumber and muskmelon are the most seriously affected host plants in the United States. Other hosts are watermelon, squash, pumpkin, gourd, wild cucumber, star cucumber, and several other species of the Cucurbitaceae (1).

Symptoms. Spots on the leaves are yellow on the upper side while on the lower side the purplish mildew appears when the humidity is high (Fig. 58). On cucumbers the affected leaves may die, and the injury to

the plant as a whole may be great enough to cause severe stunting and death. On muskmelon the spots turn from yellow to reddish brown in color, and although this host is not quite so susceptible as cucumber, plants may be killed. If plants do not die, either the fruits do not mature, or if they ripen, they do not have an attractive flavor.

The Causal Organism. *Pseudoperonospora cubensis* (Berk. & Curt.) Rostow., 1903.

Synonymy:

Peronospora cubensis Berk. & Curt., 1868.
Plasmopara cubensis (Berk. & Curt.) Humph., 1891.
Plasmopara (*Peronoplasmopara*) *cubensis* (Berk. & Curt.) Berl., 1901.
Peronospora cubensis var. *atra* Zimm., 1902.
Peronoplasmopara cubensis (Berk. & Curt.) Clinton, 1904.
Pseudoperonospora cubensis var. *tweriensis* Rostow., 1930.

The mycelium is coenocytic and intercellular and gives rise to small ovate intracellular haustoria, which sometimes develop finger-like branches. Sporangiophores arise in groups of one to five through the stomata. The upper third of the sporangiophore is branched; the branching habit is either dichotomous or between the dichotomous and monopodial, resembling that of various species of *Peronospora* more closely than that of *Plasmopara*. The sporiferous tips on which the sporangia are borne are subacute. The latter are grayish to olivaceous purple, ovoid to ellipsoidal, thin-walled, and with a papilla at the distal end. They measure 14 to 23 by 21 to 39 μ. The sporangia germinate by the production of biciliate zoospores, which are 10 to 13 μ in diameter after encystment. Rostowzew claimed to have found oospores, but this was doubted by Clinton, who like others, failed to find them. The genus was set up on a type which produced no sexual stage, but other species with oospores have been assigned to the genus since its formation.

Disease Cycle. Penetration occurs through stomata. Since no overwintering spores have been found and since cucurbits do not live as perennials in regions with severe winters, the source of primary inoculation is from infected plants growing in greenhouses or from sporangia carried northward from tropical or subtropical regions. The latter source is now known to be the most important in the United States. Beginning in Florida or Cuba, where cucurbits may be affected throughout the year, inoculum is carried northward, and new loci are established wherever a susceptible host and favorable climate coincide. The disease thus appears in successive northern locations, depending upon the weather, until it reaches New England. In some years it is checked en route by unfavorable conditions, and more northerly regions may escape or establishment of mildew may

be delayed. In the Atlantic seaboard states a reporting service conducted through the Plant Disease Survey serves to warn areas of the progress of the disease to the south and permits preparation for and timing of application of fungicides (8, 9).

While cucurbit downy mildew may appear to be favored by moist and cool environment, it is evident that moisture is the more important. Unlike some other downy-mildew fungi this fungus thrives in warm as well as cool temperatures provided fogs or dews are frequent and persistent. Sporangia germinate from about 8 to 30°C., with optima reported from 15 to 22° by various workers. About 5 hr. incubation at 18° and 100 per cent relative humidity are required for infection. Rain is not essential if heavy dews prevail. Infection occurs from about 10 to 28° with the optimum at 16 to 22°. Sporangia are formed from about 10 to 27°, with an optimum at about 15 to 19°.

Control. The use of fungicides on cucurbits has decided limitations since many forms are readily injured by Bordeaux mixture and sulfur sprays and dusts. The rapid growth of cucurbit vines requires frequent applications, and the value of the crop determines to a large extent the advisability of spraying or dusting. Insoluble-copper dusts and zinc carbamate sprays are less phytotoxic than Bordeaux mixture and copper-lime dust and are now used extensively on cucumbers and muskmelons along the Atlantic seaboard (2–4, 11).

Pioneer work in the development of mildew-resistant cucumbers was carried out by Roque (10) at the Puerto Rico Agricultural Experiment Station. After finding 150 varieties all very susceptible, he noted a Chinese variety introduced in 1933 to be highly resistant. By crossing this with local varieties, highly resistant adaptable strains were secured after selection through 10 generations. Two strains, 39 and 40, were distributed in 1942. The Puerto Rico strains were used as parents in a breeding program under way in North Carolina (6, 7). Ivanoff (5) working in Texas found four muskmelon varieties of West Indian origin to be highly resistant to mildew. By crossing with the desirable shipping type he secured a resistant variety, which was introducted as Texas Resistant No. 1 about 1945.

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DOWNY MILDEW OF LETTUCE

The downy mildew of lettuce was first described in Europe by Regel in 1843. It was recorded in the United States in 1875. It is a common disease throughout the world where lettuce is grown out of doors and under glass. It may be very destructive since the affected leaves are unsalable and trimming costs for packing and shipping are increased. It may increase on head lettuce in shipment under refrigeration, and its damage is magnified by the invasion of secondary organisms, both fungus and bacterial.

The pathogen has a large host range among economic and wild composites. Like many downy mildews, however, it is made up of a number of physiologic races, some of which do not affect lettuce.

Symptoms. Pale yellow areas appear on the upper sides of leaves and enlarge up to an inch in diameter. Opposite the spot on the lower side of the leaf the downy mildew, consisting of sporangiophores and sporangia, appears. The lesions may coalesce, and as they age, they turn brown. The color may be due in part to secondary invaders. In head lettuce the head leaves become infected, and as the lesions enlarge and discolor, they give an unsightly appearance. The growth of plants affected early is impaired, while heavy losses in mature plants are entailed from excessive trimming and reduced market value.

The Causal Organism. *Bremia lactucae* Regel, 1843.

Synonymy:

Botrytis ganglioniformis Berk., 1846.
Peronospora ganglioniformis DBy., 1863.

The mycelium is intercellular with globose haustoria. The sporangiophores are dichotomously branched. The sporiferous tip of the branch is broadened into a saucer-like disk, around the edge of which arise several

short spore-bearing sterigmata (Fig. 59). The disk-like sporiferous tip is characteristic of the genus. The sporangium is ovate, hyaline, and has a papilla at the distal end. The average size is about 17.5 by 18.5 μ. Germination takes place by a germ tube or by the formation of zoospores, which are hyaline, globular, biciliate, and about 4.2 μ in diameter (5).

Ling and Tai (4) have set up several forms within the species which differ in host range as follows: *B. lactucae* Regel on lettuce and *Lactuca indica; B. lactucae* f. *chinensis* Ling & Tai on *L. chinensis; B. lactucae* f. *sonchicola* (Schlecht.) Ling & Tai on *Sonchus oleraceus* L.; *B. lactucae* f.

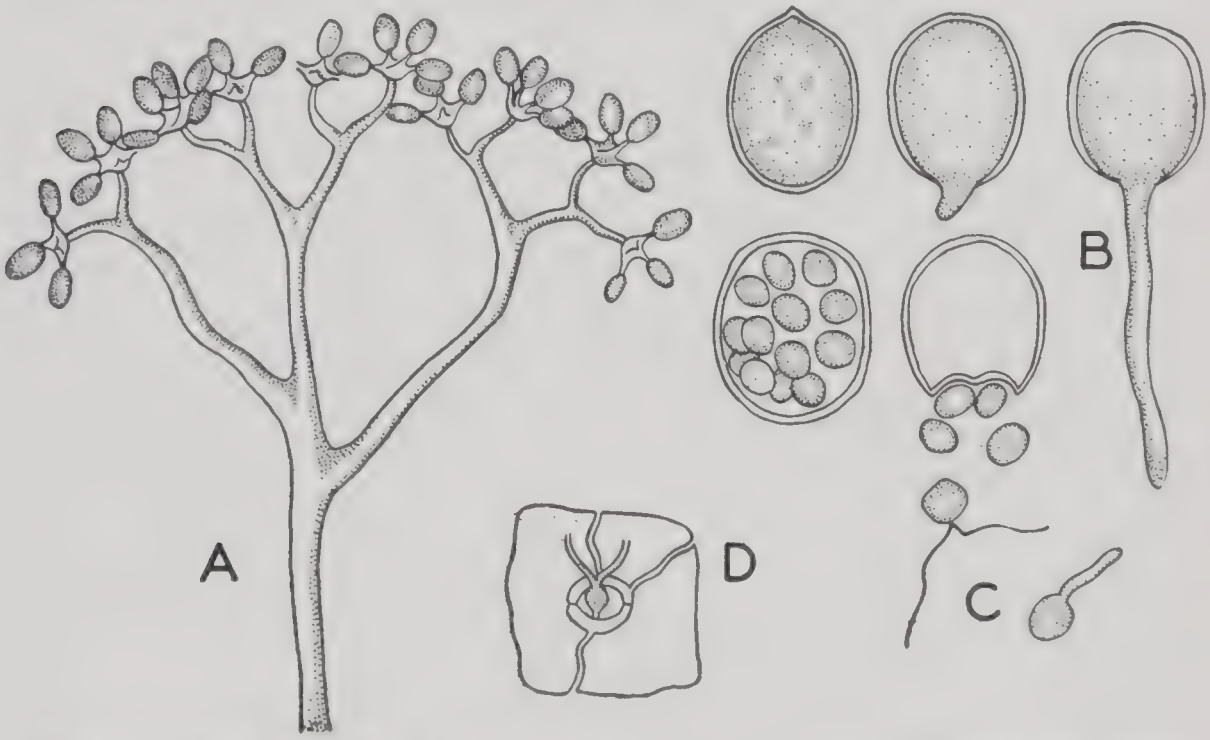

FIG. 59. *Bremia lactucae*. *A*, sporangiophore and sporangia; *B*, direct and indirect germination of sporangia; *C*, zoospore and germination of encysted zoospore; *D*, stomatal penetration by latter. (*After Milbrath.*)

taraxaci (Ito & Tokunaga) Ling & Tai on *Taraxacum mongolicum; B. lactucae* f. *ovata* (Saw.) Ling & Tai on *Crepis japonicus*. Jagger and Whitaker (3) defined five physiologic races on lettuce and suggested the possibility of one or two more.

Disease Cycle. Since lettuce is commonly grown in intensive culture, the chief means of overwintering and oversummering is the oospore stage. Ling and Tai (4) suggest carry-over in infected seeds. Local dissemination is by air-borne sporangia. Milbraith (5) noted that sporangia produced during cool weather formed zoospores more readily than those produced in warm summer temperatures. Yarwood (9) showed that sporangia are formed chiefly at night, and, as in some other downy mildews, alternation of light and darkness as well as humidity induces sporulation. Sporangia

germinate over a range from 1 to 19°C., the optimum being about 10°. Infection occurs through stomata, the optimum temperature for invasion and for fructification being 15 to 17°.

The optimum temperature for germination and infection being relatively low, the disease is typically a cool-weather one. Since high humidity is also essential for germination and infection, plenty of fog and dew are other requisites. Since lettuce is favored by relatively cool weather, the optima for host and pathogen are not far apart.

Varietal Resistance. Wide differences between varieties in the percentage of natural infection have been noted by many workers. Jagger (2) was the first to carry out an extensive breeding program for the development of resistant varieties in southern California. The favorite shipping variety, New York, was very susceptible to mildew. By crossing with a resistant Cos variety highly resistant lines of New York type were secured. Hitherto unobserved physiological races appeared, however, to which the new strains were susceptible. The control of the disease by this means has not been completely successful. Jagger and Whitaker (3) found resistance to one of the physiological races to be controlled by a single dominant gene.

Control. In glasshouse culture, spraying of the young plants with a copper fungicide is of some value (1). Regulation of humidity and temperature to keep leaf surfaces dry and to keep the temperature above the optimum for spore germination is helpful. By screening of strains and varieties some may be found which are at least temporarily resistant in a given situation. The build-up of pathogenic races should not be overlooked as a possibility.

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RHIZOPUS SOFT ROT OF SWEET POTATO

This disease was first reported on sweet potato in New Jersey by Halsted in 1890. It is generally considered to be the most destructive disease of that crop in storage. The same fungi which incite the rot of sweet potato affect most other vegetables and many fruits when conditions are favorable for infection and disease development.

Fig. 60. *Rhizopus* soft rot of sweet potato. (*Courtesy of U.S. Department of Agriculture.*)

Symptoms. The disease begins as a soft, watery rot which progresses rapidly in the fleshy tissue. A sweet-potato root may be completely rotted within 4 or 5 days after infection is first visible. The fleshy tissue becomes soft and stringy, and water exudes readily when the skin is broken. The tissue turns brownish in color, and a mild odor is noticeable. As the skin is broken, the grayish sporangiophores and mycelium and dark-colored sporangia develop very rapidly, producing a coarse whiskery extramatrical growth which is very characteristic of the disease not only on sweet potato but on other vegetables and fruits which may become infected (Fig. 60). The affected tissue sometimes desiccates rapidly and gives the disease the appearance of dry rot. In some cases the tissue may rot in zones adjoining several points of infection on a root and dry out rapidly causing a series of decayed rings. This phase is referred to as ring rot.

The Causal Organism. *Rhizopus nigricans* Ehr., 1820.

Synonymy:

Mucor mucedo L., 1753.
Ascophora mucedo Tode, 1790.
Mucor stolonifer Ehr., 1818.
Mucor ascophorus Lk., 1824.

The coenocytic mycelium has prominent aerial branches, referred to as *stolons*, which arise from nutritive hyphae in the substrate. As they reach 1 to 2 cm. in height, they bend over to form tufts of branched rhizoids at points of contact with the host. Upright branches arise in fascicles at the point of origin of the rhizoids, and each becomes a sporangiophore bearing a terminal sporangium, except for one branch, which becomes the advancing stolon. The sporangium arises as a swelling of the tip of the sporangiophore. There is extensive streaming of the protoplasm as the sporangium enlarges, and the former becomes denser toward the periphery. The columella is a spherical projection of the sporangiophore into the base of the sporangium which persists after the sporangial wall has broken to release the spores. It starts with the formation in the lower portion of the sporangium of a layer of vacuoles, globular at first, but assuming disk-like shapes and joining to form a cleft in the protoplasm. In the meantime, a furrow which has begun to form near the base of the sporangium rises to join the cleft of vacuoles. This line develops into a columella wall. Meanwhile spore formation has proceeded with the appearance of furrows in the multinucleate protoplasm starting at the periphery and at the columella (Fig. 61). By means of an intricate system of branching and fusing the entire protoplasm is divided into small portions, which are angular at first and round up in irregular arrangement to form the sporangiospores. Each spore contains two to six nuclei, which do not divide as the spore matures. Ripe spores are mostly ovoid with walls marked externally with longitudinal ridges. The spores are at first embedded in a jelly-like matrix secreted from the walls. They are not discharged forcibly when the sporangial wall breaks and sometimes cling in clusters to the columella. They are readily borne by air currents, and each germinates by a germ tube which branches promptly and profusely (17).

In the formation of the zygospore, lateral branches from two separate hyphae arise, come in contact, and each swells to form a progametangium and delimits from the latter a terminal cell, the gametangium (Fig. 61). The gametangia, which are practically indistinguishable in size and shape, fuse and give rise to the zygote. Some nuclei from the two gametangia become paired, and many degenerate. The paired nuclei fuse before the zygospore becomes dormant, and meiosis takes place upon germination. The zygospore, as it matures, is provided with a black, thick, several-layered wall. Germination consists in the formation of a short germ tube with a terminal sporangium known as the germ sporangium (3, 4).

There are some nine species of *Rhizopus* which may incite similar symptoms on sweet potato, some requiring a higher temperature than *R. nigricans* for optimum development. The latter species is by far the most common

and usually outstrips others which may occur on the same substrate (7–10).

Many students of this group of fungi have noted that zygospore formation is erratic. The fundamental reason was given by the discovery by Blakeslee (1, 2), reported in 1904, that the formation of zygospores in *R.*

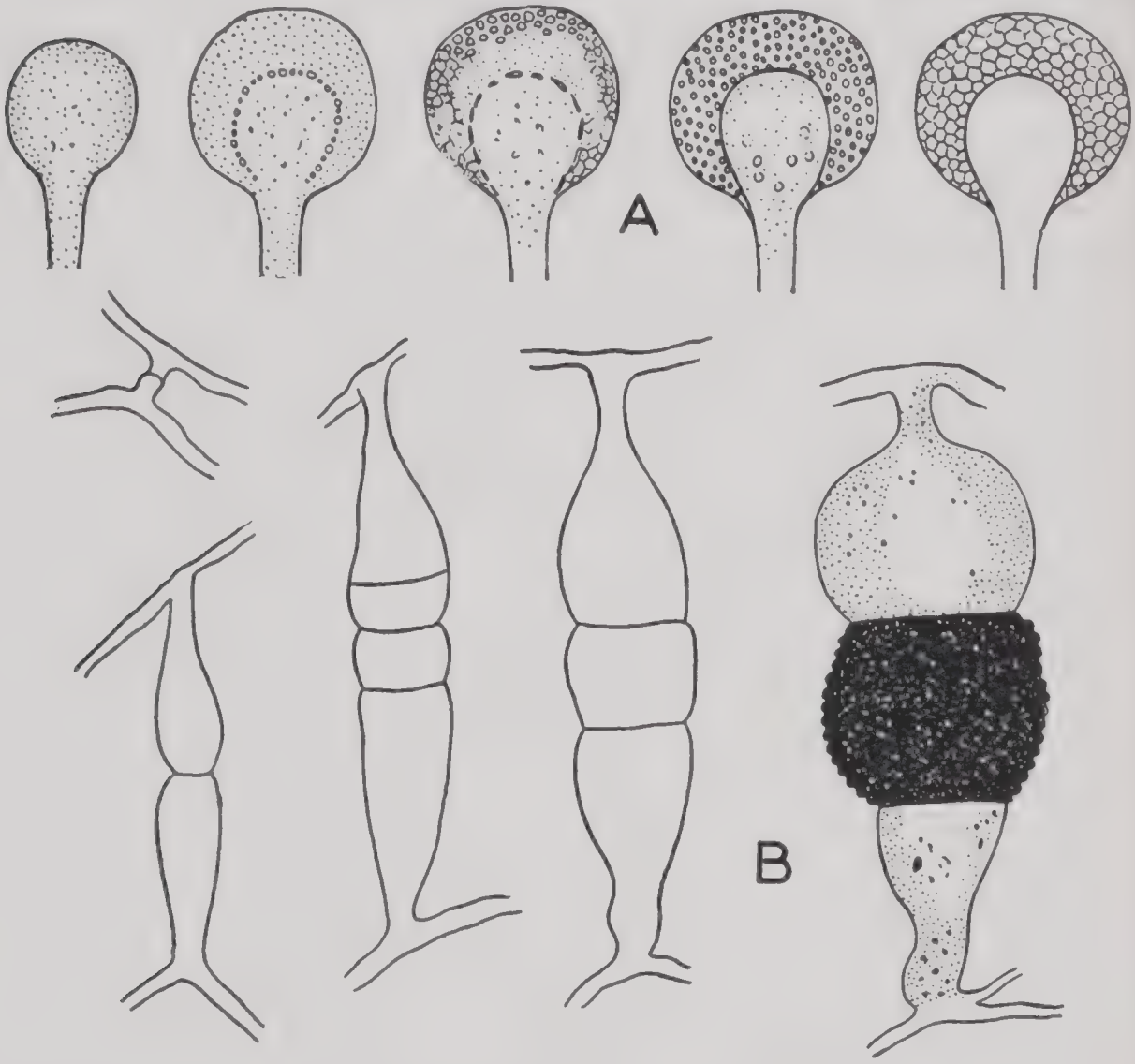

FIG. 61. *Rhizopus nigricans.* *A*, development of sporangiophore, sporangium, and sporangiospores; *B*, development of zygote. (*A after Schwarze; B after De Bary.*)

nigricans was dependent upon the mating of compatible strains which he referred to as + and −. Since the gametangia were similar, it was not possible to designate them as male and female on the basis of size, as is the case in the Oomycetes. Blakeslee regarded the reaction as an indication of sexuality. This phenomenon is called *heterothallism*, in contrast with *homothallism*, in which male and female organs arise from the same thallus. Further study showed that some species of *Rhizopus* were homothallic, and throughout the Mucorales forms differ in this respect. Fur-

ther reference to heterothallism in relation to sexuality in fungi is made in Chap. 14.

Disease Cycle. The zygospores serve as resting spores, and the sporangiospores may also survive for many months. The fungus is such a good saprophyte that it no doubt subsists on crop debris between crop seasons. Actively growing tissue is seldom affected, and the parasitism of the various species of *Rhizopus* is confined to more or less dormant storage organs of fruits and vegetables. These species are strictly wound parasites. Once infection is established, decay is brought about by the action of propectinase in advance. The type of parasitism, then, is very similar to that of the bacterial-soft-rot organism.

Infection is influenced by environment and host reaction. Harter and associates showed that when cut surfaces of sweet potato in a moist chamber at optimum temperature for decay were smeared with spores and hyphae of *R. nigricans*, infection seldom occurred. Placing the fungus in a "well" made in the sweet-potato root did not ensure infection. However, if the fungus was allowed to grow on a vegetable decoction for 24 hr. and the suspension of fungus in the substrate was placed in the "well," infection occurred regularly and promptly. One of the reasons for failure of the inoculation on a freshly cut surface in a moist chamber is that the host tissue responds rapidly in cork-cambium formation, which excludes the fungus. When the enzyme is placed in the well with the fungus, infection proceeds before cambium activity becomes effective. In the storehouse, wounds commonly heal over or, more often, seal over in some way so as to exclude the fungus.

All species of *Rhizopus* studied produced propectinase in culture, but there was no correlation between parasitism and degree of production of the enzyme. In fact, one species which produced propectinase in greatest abundance was not pathogenic on sweet potato, while *R. nigricans*, which was among the lowest of 11 species in enzyme production, was the most aggressive pathogen. Thus, while enzyme production is a factor in pathogenesis of *Rhizopus*, there are other factors involved which are not as yet understood.

The minimum temperature for growth of *R. nigricans* on culture medium is about 6°; optimum, 23 to 26°; maximum about 31°C. For sporulation the minimum is about 10°; optimum, 23 to 28°; maximum about 30°. For spore germination the minimum is about 1°; optimum 26 to 28°; maximum about 30°. Germination occurs in about 5 hr. at 20 to 30°; in about 15 hr. at 15° and 33°. The cardinal temperatures for decay of sweet-potato roots are minimum, 3 to 7°; optimum, 15.5 to 23°; maximum, 29 to 34°. Isolates differ somewhat in temperature limits and optima (13–15)).

Saturated atmosphere is not necessary for infection, and after the fungus

has become established, decay may proceed at low relative humidity. The optimum relative humidity at 23°C. is 75 to 84 per cent, and the percentage of infection decreased when the humidity was raised or lowered, with very little infection at 93 to 99 per cent. Inoculated halved roots held at 51 to 73 per cent relative humidity for 4 to 12 days became infected when placed after that period at 84 to 95 per cent. One of the interacting factors is the effect of environment on the protective cork formation. High relative humidity (95 to 100 per cent) is most favorable for wound cork formation in the sweet potato. It occurs readily from about 19 to 33° with maximum activity at 33°. As growth of the pathogen increases with temperature, it is increasingly offset by increase in protective action of the host tissue. The optimum for disease development is lower, therefore, than it might be otherwise. This is a good example of the effect of environmental factors upon the interaction of host and pathogen, a subject which is discussed further in Chap. 13.

Control. Sanitation of the storage rooms is important. Debris should be removed promptly and walls, floors, and containers disinfested before the crop is stored.

Since bruises become the portal of invasion, care in harvesting and handling is essential.

The most direct control measure is regulation of the storehouse. When the crop is harvested and stored, the common procedure is to raise the temperature of the house by artificial heating to 28 to 32°C. for 2 weeks. During this period corking or sealing of wounds is hastened, and penetration other than that which has already occurred is prevented. The storage temperature is then lowered to a level which will reduce the rate of decay which has started from infection by *R. nigricans* or other pathogens. However, storage below 5°, though desirable from the standpoint of reduction in decay, is not practicable because of the tendency for nonparasitic internal necrosis to develop in sweet-potato roots.

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CHAPTER 7

DISEASES INCITED BY FUNGI IMPERFECTI

The Fungi Imperfecti, or Deuteromycetes, more familiarly known as "the Imperfect Fungi" or simply as "the Imperfects," are a more or less artificial assemblage of some thousands of species which do not have a perfect stage but otherwise, in structure and in reproductive methods, resemble fungi of the higher classes, Ascomycetes (Chap. 8), and Basidiomycetes (Chap. 9). Their affinities appear to be mainly with the former class. As indicated in more detail below, presence or absence of a fruiting body, type of fructification if one is found, the character of the spore if spores are produced, and mycelial peculiarities are among the characters employed in classifying these forms. Perfect stages have been found later for many species originally in this class, and these have then been removed to the proper genus in another class.

Many plant diseases are incited by members of the Fungi Imperfecti, and therefore they are of great importance to plant pathology although they hold, as a rule, less interest for the systematic mycologist. It will be seen that in some of the diseases incited by Ascomycetes and Basidiomycetes treated in later chapters, the imperfect stage is the important one in so far as disease production is concerned, and that many of these forms may persist indefinitely without occurrence of the perfect stage.

The class is divided into four orders based upon the type of, or lack of fructification. These orders are subdivided into families on the basis of certain characters of the fruiting bodies. Subfamilies and genera are based chiefly upon the color, shape, and septation of conidia.

The orders, families, and certain of the most important genera are described briefly:

Moniliales (Hyphales). Conidiophores formed singly, in fascicles (coremia), or in cushion-shaped structures (sporodochia).

Moniliaceae. Sporodochia and coremia absent; hyphae and conidiophores (at least in the earlier stages) and the conidia usually hyaline or bright colored.

Monilia. Conidiophores short and conidia produced in branching chains.

M. fructigena Pers. is the imperfect stage of *Sclerotinia fructigena* (Pers.) Aderh. & Ruhl., one of the brown-rot fungi.

Oidium. Branched hyphae on the surface of the living host giving rise to erect, unbranched, short conidiophores bearing ovoid conidia in chains. Most imperfect stages of the powdery-mildew fungi (Erysiphaceae) belong to this genus.

Aspergillus. Conidiophores, erect, unbranched, with a terminal swelling;

conidia in long chains borne on short but clearly defined sterigmata, which often nearly cover the entire bulbous tip of the conidiophore.

Penicillium. Erect conidiophores which become penicillately branched at the tip and bear chains of conidia.

Botrytis. Erect conidiophores bearing conidia on rounded tips of branches.

B. allii Munn incites neck rot of onion.

Dematiaceae. Hyphae, conidiophores, and conidia typically brown or dark.

Fusicladium. Short, erect, straight, sparingly septate conidiophores bearing ovoid, continuous, or one-septate conidia acrogenously.

F. dendriticum (Wallr.) Fckl. is the imperfect stage of *Venturia inaequalis* (Cke.) Wint., incitant of apple scab.

Alternaria. Short, erect conidiophores bearing dark, muriform, beaked conidia in chains.

A. solani (Ell. & G. Martin) L. R. Jones & Grout, incites early blight of potato and tomato.

Stilbaceae. Conidiophores aggregated into a cylindric fascicle or coremium.

Graphium. Dark coremia with hyaline, one-celled conidia.

G. ulmi Schwarz is the imperfect stage of *Ceratostomella ulmi* Buisman, incitant of the Dutch elm disease.

Tuberculariaceae. Conidiophores compacted to form a sporodochium.

Fusarium. Macroconidia septate, fusoid, hyaline, borne terminally on branched conidiophores; microconidia hyaline, mostly continuous.

F. oxysporum f. *conglutinans* (Wr.) Snyder & Hansen, incitant of cabbage yellows.

F. oxysporum f. *lycopersici* (Sacc.) Snyder & Hansen, incitant of *Fusarium* wilt of tomato.

Melanconiales. Conidiophores aggregated in a palisade layer on the surface of a thin stroma usually forming below and rupturing the host cuticle or epidermis; the fruiting body is known as an acervulus. Melanconiaceae is the only family.

Colletotrichum. Conidia continuous, hyaline, oblong to fusoid; setae scattered throughout acervulus.

C. circinans (Berk.) Vogl., incitant of onion smudge.

C. lindemuthianum (Sacc. & Magn.) Briosi & Cav., incitant of bean anthracnose.

Gloeosporium. Similar to *Colletotrichum* except for absence of setae.

G. fructigenum Berk. is the imperfect stage of *Glomerella cingulata* (Ston.) Spauld. & Schrenk, incitant of bitter rot of apple.

Sphaeropsidales (Phomales). Conidia (pycnospores) produced in variously shaped semiclosed bodies known as pycnidia.

Sphaerioidaceae (Phomaceae). Pycnidia brown or black, usually flask-shaped, and with a small apical opening (ostiole).

Phoma. Conidia continuous, hyaline.

P. lingam (Fr.) Desm., incitant of blackleg of crucifers.

Ascochyta. Conidia typically one-septate, hyaline.

A. pinodes L. K. Jones is the imperfect stage of *Mycosphaerella pinodes* (Berk. & Blox.) Stone, incitant of *Mycosphaerella* blight of pea.

Diplodia. Conidia, dark-colored, typically one-septate.

D. zeae (Schw.) Lév., incitant of *Diplodia* disease of corn.

Septoria. Conidia hyaline, continuous or septate, elongate-fusoid, rod-shaped, or filiform.

S. apii (Br. & Cav.) Chester and *S. apii-graveolentis* Dorogen, incitants of late blight of celery.

Nectrioidaceae. Pycnidia similar to those of Phomaceae but hyaline or bright colored.

Leptostromataceae. Pycnidia shield-shaped.

Excipulaceae. Pycnidia with broad ostioles or irregular openings; cupulate, discoid, or hysteroid.

Mycelia Sterilia. No conidia formed.

Sclerotium. Sclerotia variable in size, shape, and color, and connected by mycelial threads.

S. cepivorum Berk., incitant of white rot of onion.

Rhizoctonia. Sclerotia embedded in mycelial matrix.

R. solani Kühn is the imperfect stage of *Pellicularia filamentosa* (Pat.) Rogers, incitant of the *Rhizoctonia* diseases of many plants.

GRAY-MOLD NECK ROT OF ONION

The first description of neck rot of onion was published by Sorauer in Germany in 1876. The disease was reported in the United States in 1890 and in England in 1894. It is now known in many countries and is one of the major bulb-destroying diseases of this crop. In 1925, Walker (6, 8) distinguished three species of *Botrytis*, each of which may incite a neck rot of onion bulbs, the respective diseases differing somewhat in symptoms. They were given distinctive names as follows: gray-mold neck rot (*Botrytis allii* Munn); mycelial neck rot (*B. byssoidea* J. C. Walker); small-sclerotial neck rot (*B. squamosa* J. C. Walker). The most common of the three is gray-mold neck rot, which will be considered here. The pathogen also infects shallot (*Allium ascalonicum* L.) and garlic (*A. sativum* L.).

Symptoms. The disease is found most commonly upon the bulbs after harvest, infection usually taking place through the neck tissue but occasionally through the stem end or through wounds elsewhere. The first sign is the softening of the affected scale tissue, which takes on a sunken, cooked appearance. This remains characteristic of the advancing region of decay, which is separated from healthy tissue by a definite margin. Since the effect upon host cells is produced somewhat in advance of the hyphae of the pathogen, there is usually little evidence of mycelium in the outer zone of decaying tissue. As the mycelium increases in the older diseased area, the tissue becomes grayish in color, and later a dense

grayish mycelial mat often develops upon the surface of the scales. Under moderately humid conditions conidial production is prompt. A dense layer of gray mold appears, which consists of comparatively short conidiophores and myriads of conidia (Fig. 62).

The disease progresses most rapidly down the scales which have been originally infected at the neck. The spread from scale to scale proceeds somewhat more slowly. In a cross section of a partially rotted bulb, at the advance margin of the disease the parenchyma tissue has a distinctly water-soaked appearance. A few millimeters back from this point a grayish tinge and a slight shrinkage in the decaying tissue become evident, and still farther back shrinkage and mycelial development increase. In connection with the older decayed tissue sclerotia appear, first as whitish

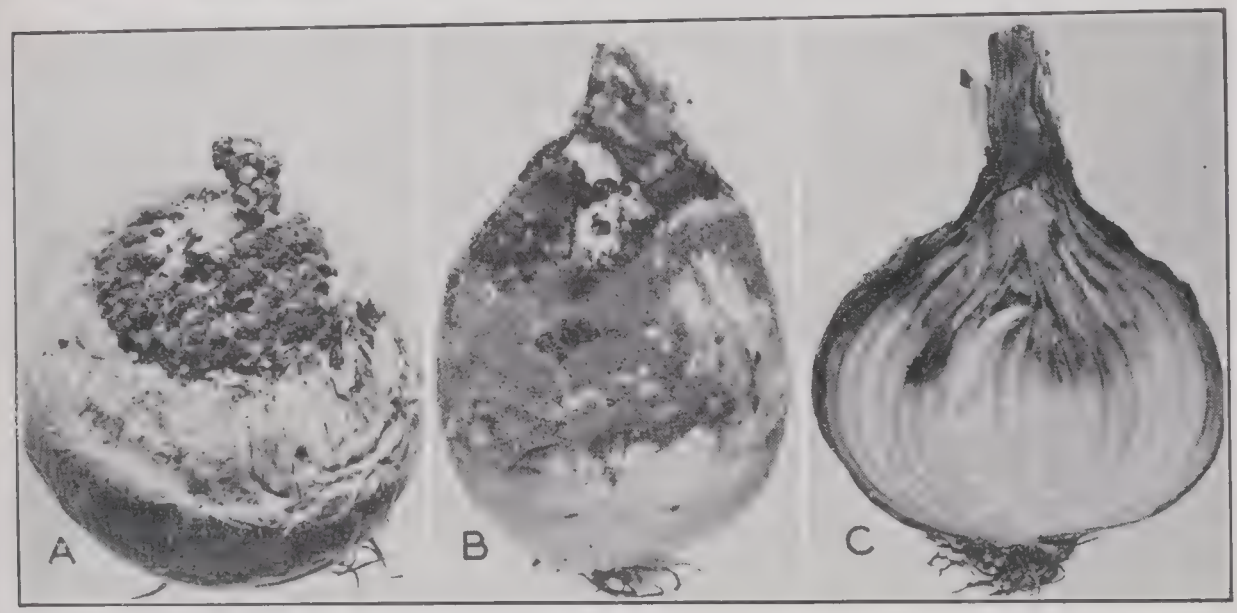

FIG. 62. *A*, mycelial neck rot of onion. *B*, *C*, gray-mold neck rot of onion.

compacted masses of mycelium, which become darker with age until they assume the appearance of hard, black, rounded, kernel-like bodies, spherical, oblong, or irregular, and varying from 1 to 5 mm. in length. They form usually on the outer surface of the scale or are slightly embedded in the diseased host tissue. While the host tissue in the beginning of its decay is somewhat watery, it desiccates rather promptly. The moisture which is released is often sufficient to stimulate premature sprouting. The older decayed bulb presents the appearance of a "mummy." While the pathogen is primarily a parasite on dormant fleshy scales, it may cause small white necrotic lesions on seedstalks. They may induce sufficient damage to cause blasting or reduce the quality of the seed (1, 11).

The Causal Organism. *Botrytis allii* Munn, 1917. The mycelium is septate, branching, hyaline when young, taking on a slight tinge of color with age. Aerial hyphae take on a smoky-gray cast en masse, but

only in the conidiophores do the cell walls take on a deep brown color. Appressoria, or holdfasts, are commonly formed by repeated branching of hyphae which come in contact with the host surface, and they form abundantly in contact with glass containers of pure cultures. In the appressoria the cytoplasm appears to be denser than in other hyphae, and the cell walls darken somewhat. Sclerotia form as velvety, white, dense masses of mycelium in or upon the decayed host tissue. The surface assumes a waxy appearance and gradually darkens through light brown to darker shades until a hard, black, kernel-like mass results. The mature sclerotia are variously shaped; they are rounded on the upper surface and are either flat or concavely depressed on the lower side when attached to the outer surface of the onion scale. They vary from 1 to 5, mostly 2 to 4 mm., in diameter. They may aggregate into a large crusty mass. In cross section they show a pseudoparenchymatous structure consisting of closely packed mycelial threads. The several outer layers consist of dark walled cells which give the external black color to the sclerotium; the remaining interior is made up of hyaline cells. Under suitable conditions sclerotia germinate by sending out mycelial branches or by the direct production of conidiophores upon the surface. Sclerotia of this general type are found in many species of *Botrytis* and in the ascomycetous genus *Sclerotinia*.

The conidiophores of *Botrytis* are on the surface of the host substrate. They arise as branches of the mycelium, remaining hyaline at the growing tip but becoming dark-walled with age (Fig. 63). The growing tip sends out several side branches, each of which becomes rather swollen at the end and gives rise to many sterigmata. The sterigmata swell gradually at their tips to form the conidia. While the spores are enlarging, they are not readily removed, but as soon as they are mature, they become detached from the short sterigmata. The sporiferous branches then collapse as far back as the main conidiophore, which again grows forward at the tip, branches, and repeats the process. The points at which the sporiferous branches grew out and later collapsed are shown by slight warty protuberances. The indeterminate sporiferous habit results in very profuse conidial formation. The conidia are disseminated very readily by air currents, and though they do not disperse in water readily, they germinate promptly when once immersed. Conidia are oblong to elliptical, hyaline, nonseptate, 6 to 16 by 4 to 8 μ, mostly 7 to 11 by 5 to 6 μ. Microconidia are not common; they are globose, hyaline, about 3 μ in diameter and are borne on short hyaline conidiophores.

Variability of the fungus is discussed by Owen *et al.* (5).

Disease Cycle. The organism overwinters as sclerotia in the soil and in bulbs. Decayed bulbs dumped from warehouses may be a source of

inoculum. Since the growing plant is seldom affected, the fungus maintains a saprophytic existence during the growing season. It may be found on dead sloughed scales in moist weather previous to harvest. Parasitic existence begins after infection occurs during the harvest period. Neck rot is an extremely spasmodic disease, its greatest epidemic development occurring when cool, moist weather prevails for some days previous

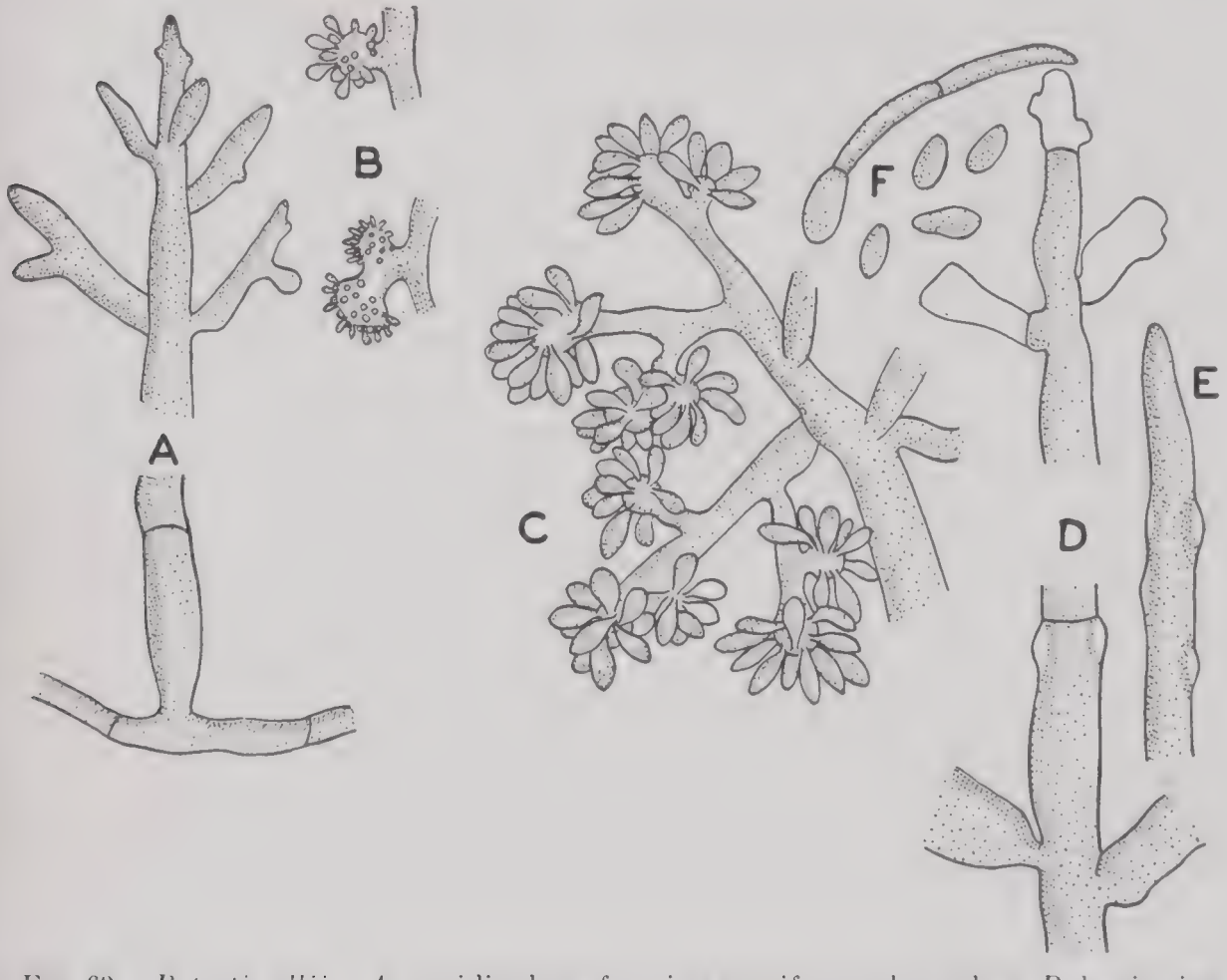

FIG. 63. *Botrytis allii*. *A*, conidiophore forming sporiferous branches; *B*, beginning of spore formation; *C*, nearly mature conidia at extremities of the branches; *D*, collapse and disintegration of sporiferous branches back to the main axis after dehiscence of conidia; *E*, new growth from apex of the main axis to form more sporiferous branches and conidia; markings of old branches remain; *F*, conidia and germinating conidium.

to and more or less frequently after harvest (2, 4, 8). Warm dry harvest weather is unfavorable. While conidial production, germination, and mycelial growth occur over a wide temperature range, infection and post-infection decay of bulbs are clearly favored by temperatures between 15 and 20°C.

The succulence of the neck tissue at harvest time is important in determining the amount of infection. If bulbs mature well during dry weather before harvest occurs, the chances of infection are greatly reduced. Even

after incipient infection occurs, rapid drying out of the decayed neck tissue may effectively check any further progress of the disease (7).

Munn (3) has shown that the cytolytic action on the host tissue in advance of the hyphae is brought about by propectinase produced by the fungus. The "action in advance," then, is somewhat similar to that described in bacterial soft rot. In the bacterial soft rot of onion, however, the softening and plasmolysis of the tissue is much more rapid and extensive than in neck rot, and a sulfurous odor is characteristic of the former disease.

Varietal Resistance. Sorauer early observed that white varieties of onion were more frequently affected than colored varieties. This has been observed many times since. It has been shown by Walker and associates (2, 4, 9) that certain phenolic substances in the dry, outer, colored scales are antibiotic to *Botrytis allii* and that when the fungus encounters this material at the neck, infection may be precluded. Since these materials are contained chiefly in the outer scales and only in the outer epidermal cells of the succulent scales, infection may occur when succulent neck tissue is exposed at harvest. Once the fungus has invaded the succulent tissue, bulbs of colored and white varieties are equally susceptible. Thus, while colored varieties are generally less affected, there may be circumstances in which they become very severely diseased. There is evidence that other antibiotic substances in the fleshy tissue affect the progress of the pathogen. Hatfield *et al.* (2) and Owen *et al.* (4) found that mildly pungent varieties were significantly more affected than strongly pungent varieties regardless of color. The relation of color and pungency in onion to disease resistance is discussed more fully under onion smudge.

Control. One of the control measures, of course, is to grow colored rather than white varieties, where practicable. Since onion crops are commonly grown in very close succession, crop rotation is not practicable, and it might do little good with a pathogen so readily dispersed by air currents. The chief direct control practice is to allow tops to mature well before harvest; keep harvest bruises such as those which occur in mechanical toppers to a minimum; provide aeration and as thorough curing as possible. Artificial curing of bulbs at harvest by heating in a rapidly moving dry air current at 37 to 48°C., sufficient to dry the necks thoroughly, will check neck rot if it has not advanced beyond the incipient stage (7). In commercial practice this procedure has been used, in only a limited way, on white-onion sets. The best storage conditions for table-stock onions and for onion sets are 0°C. and relative humidity of about 64 per cent (10). Artificial ventilation of onion storage houses to keep the relative humidity from approaching saturation is desirable.

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THE FUSARIAL DISEASES

The genus *Fusarium* comprises many species and many forms within species. Macroconidia are hyaline, fusoid, sometimes pedicellate; one- to several-septate; borne acrogenously. Conidiophores are branched and may be scattered, or they may rise in tufts covered with a more or less slimy mass of conidia to form the tuberculate fruiting body known as the **sporodochium.** The latter may have a flat wart-like base known as the **plectenchyma.** Some species produce macroconidia sparsely. Microconidia are hyaline and one- to four-celled. Chlamydospores, unicellular or one-septate, are thick-walled and terminal or intercalary. Plectenchymatic sclerotia may be formed. Spore masses, sclerotia, and mycelium are commonly colored.

Wollenweber (5, 6) divided the genus into several sections, *e.g.*, *Elegans*, *Martiella*, *Discolor*, *Gibbosum*, *Roseum*, *Ventricosum*. His system of classification has been followed widely since about 1913. Snyder and Hansen (2–4) in 1940 pointed out that variability within species and single-spore lines was great enough to invalidate many of the species distinctions set up in the Wollenweber system. They proposed that the number of species be reduced to those that can be reliably described on the basis of consistent morphological differences and that biotypes within such species be designated as forms and races. Thus all species in the section *Elegans* are placed under *F. oxysporum* Schlect.; all species in the

section *Martiella* under *F. solani* (Mart.) Appel & Wr., etc. The system of Snyder and Hansen is followed in this book.

All species of *Fusarium* have a saprophytic stage. Many are only mild facultative parasites. Some are primarily decay-producing organisms on dormant vegetable organs. Others are mild root parasites. Some are primarily cortical invaders causing preemergence damping-off, foot rots, and stem cankers. The most highly developed pathogens are those contained within the species *F. oxysporum*, which invade the xylem vessels of their hosts and are referred to as the vascular fusaria.

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THE VASCULAR FUSARIAL DISEASES

There are more than a score of diseases incited by forms of *Fusarium oxysporum* Schlecht. in which the pathogen, invading mostly through the fibrous root system, is present chiefly in the large xylem elements. The symptoms usually consist of various gradations of chlorosis, stunting, vascular discoloration, and oftentimes wilt. In view of the fact that wilt is a common symptom in many of these diseases, they are often referred to as fusarial wilts. In general, the disease effects appear in advance of the fungus. Sometimes, as in pea wilt, the plant may become chlorotic and die while the fungus is confined to the lower four or five internodes of the stem. The pathogens are very successful inhabitants of the soil and after becoming established in the soil remain there almost indefinitely. Infested soil thus becomes unfit for production of the crop concerned unless resistant varieties are available.

As a rule, the form of *F. oxysporum* pathogenic on a given species is restricted in its pathogenicity to that species or to closely related ones. In some there is a phase of the disease in which the organism causes cortical decay. An example is the watermelon wilt where, under low tempera-

tures, the fungus causes preemergence damping-off. This situation is, however, an exception to the general rule. As a class, the vascular fusaria are favored by relatively high soil temperature, and in some cases, as in cabbage yellows and tomato wilt, it is possible to grow the host in cool infected soil without sufficient infection to produce symptoms.

In practically all of the fusarial wilts some individuals or varieties are found which are resistant to the pathogen, and in many the development of resistant varieties has served to control the disease.

Some of the important diseases in this group and their respective pathogens follow. Two of them (cabbage yellows and tomato wilt) are described in detail.

Tomato wilt, *F. oxysporum* f. *lycopersici* (Sacc.) Snyder & Hansen.
Cotton wilt, *F. oxysporum* f. *vasinfectum* (Atk.) Snyder & Hansen.
Watermelon wilt, *F. oxysporum* f. *niveum* (E. F. Sm.) Snyder & Hansen.
Flax wilt, *F. oxysporum* f. *lini* (Bolley) Snyder & Hansen.
Cabbage yellows, *F. oxysporum* f. *conglutinans* (Wr.) Snyder & Hansen.
Banana wilt, *F. oxysporum* f. *cubense* (E. F. Sm.) Snyder & Hansen.
Celery yellows, *F. oxysporum* f. *apii* (R. Nels. & Sherb.) Snyder & Hansen.
Pea wilt, *F. oxysporum* f. *pisi* (Linford) race 1 Snyder & Hansen.
Pea near-wilt, *F. oxysporum* f. *pisi* (Linford) race 2 Snyder & Hansen.
Muskmelon wilt, *F. oxysporum* f. *melonis* (Leach & Currence) Snyder & Hansen.
Aster wilt, *F. oxysporum* f. *callistephi* (Beach) Snyder & Hansen.
Broad-bean wilt, *F. oxysporum* f. *fabae* Yu & Fang.
Stock wilt, *F. oxysporum* f. *mathioli* Baker.

CABBAGE YELLOWS

Erwin F. Smith first called attention to this disease on cabbage in the Hudson valley in 1899. During the first decade of this century it increased rapidly in cabbage-growing areas in Ohio, northern Illinois, southern Wisconsin, and southeastern Iowa. It has been found since in many other states in the same latitude and in those farther south. It occurs as far north as Rochester, New York, and Green Bay, Wisconsin. The disease has been reported in Cuba, Trinidad, Rhodesia, and the Philippine Islands. In this country it is most serious where cabbage is grown in warm seasons. In southern states, where much of the cabbage is planted as a winter crop, the disease is of minor importance.

All members of the cabbage tribe (*Brassica oleracea* L.), including cauliflower, kale, broccoli, kohlrabi, and collard, are more or less susceptible.

Symptoms. The disease affects plants at any age. In northern states, where seed is sown in late April or early May, plants may not show signs until after transplanting. When plants are set in the field in mid-June, the disease appears 2 to 4 weeks after transplanting. The first

sign is the lifeless yellow-green color of the foliage. Sometimes the yellowing is uniform, but more often it is more intense on one side of leaf and plant, causing a lateral warping or curling of the leaves and stem (Fig. 64). The lower leaves become yellow first, and the appearance of symptoms progresses upward. As the yellowed tissue ages, it turns brown and becomes dead and brittle. Affected leaves drop prematurely, and normal growth of the plant is distinctly retarded. The vascular system becomes

FIG. 64. Cabbage yellows. Note unilateral development of affected leaves (*left*) and discoloration of vascular bundles (*center*).

yellow to dark brown. In many respects the disease resembles black rot (p. 122) and is very often confused with it. A fairly reliable point of distinction is the fact that in black rot the veins become black rather than brown and the smaller veins of diseased leaves are much more generally discolored.

The Causal Organism. *Fusarium oxysporum* f. *conglutinans* (Wr.) Snyder & Hansen, 1940.

SYNONYMY: *Fusarium conglutinans* Wr., 1913.

The fungus produces septate mycelium, which grows well on many vegetable agars. It is colorless at first, becoming cream-colored with age and finally showing ocherous strands throughout the colony. In older mycelium chlamydospores are formed (Fig. 65). The fungus produces in abundance single-celled, hyaline, ovoid to ellipsoid microconidia, 2.5 to 4 by 6 to 15 μ. Macroconidia are usually relatively rare; they are fusiform, hyaline, and mostly two- to three-septate, 3.5 to 5.5 by 25 to 33 μ. Sporodochia, pionnotes, and sclerotia are absent.

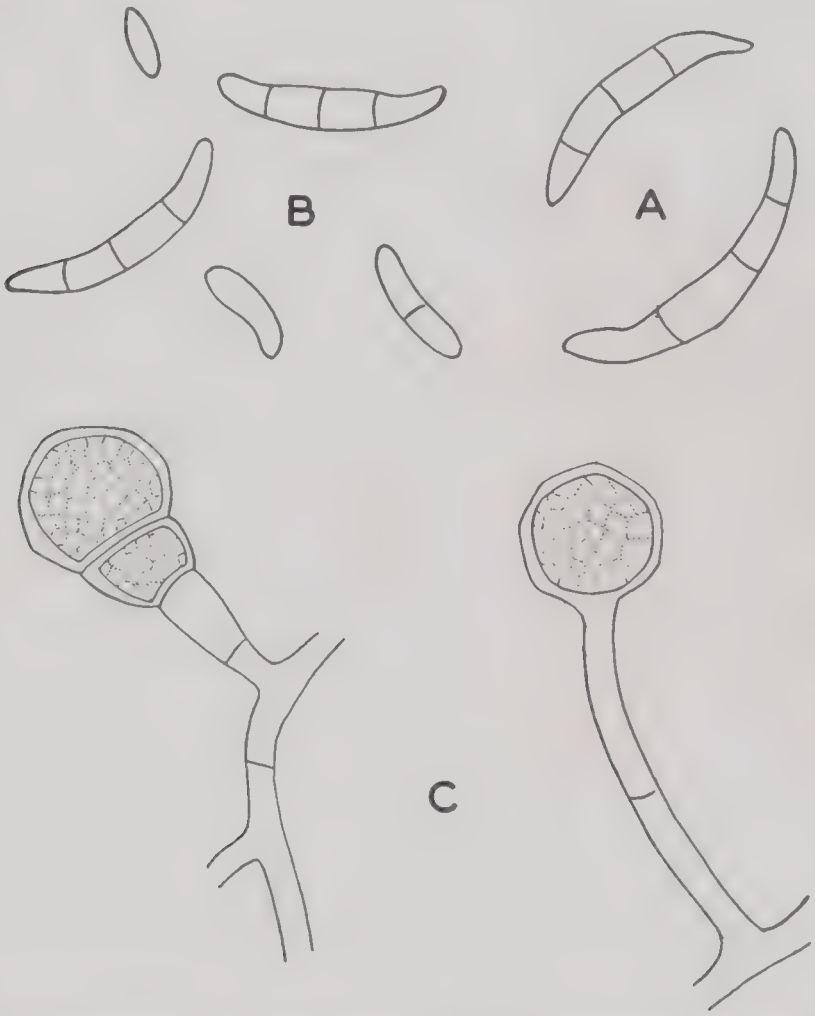

FIG. 65. The cabbage-yellows organism. *A*, *B*, macroconidia and microconidia; *C*, chlamydospores. (*After Gilman.*)

Disease Cycle. The organism is distributed over long distances in transplants and in infested soil clinging to potato seed tubers and other plant parts used for propagation. Local dissemination is by means of soil on implements, wind-borne soil, surface drainage water, and water-borne soil. The organism thrives in a variety of soil types, and once it is established, it remains viable indefinitely. Infection takes place through the root-tip region or through wounds created at transplanting. The fungus invades the root cortex with little damage to the latter and becomes established in the spiral vessels and progresses upward within the large

xylem elements (Fig. 66). Occasionally microconidia are produced in the xylem. The organism does not invade other tissue until the plant dies.

The rate of fungus growth on agar increases with temperature up to an optimum of about 28°C., beyond which point the rate decreases rapidly. The association of the disease with warm weather and its limited occurrence in cool northern regions led to an intensive study of the relation of controlled soil temperature to disease development. This was one of the first fusarial diseases studied thoroughly in Wisconsin soil-temperature tanks (Chap. 13). Gilman (7), Tisdale (14), Tims (13), Walker and Smith (20) and Blank (3, 5) have made contributions. In general, the disease curve rises and falls roughly parallel with the growth curve of the organism, which has been taken to indicate that the effect of soil temperature on disease development is expressed primarily through its effect upon the organism. Tisdale (14) established a "critical temperature" of 17°C., below which the disease did not occur when susceptible plants were grown in infested soil. Blank (3) showed later that with an extremely susceptible line the disease occurred at as low a constant soil temperature as 12°. Tisdale (14) found soil moisture to have a relatively minor influence upon the disease. Walker and Hooker (22) working in pure sand culture showed that the rate of disease development declined as the concentration of a balanced nutrient increased, and that in a solution low in potassium the rate and severity of disease development increased.

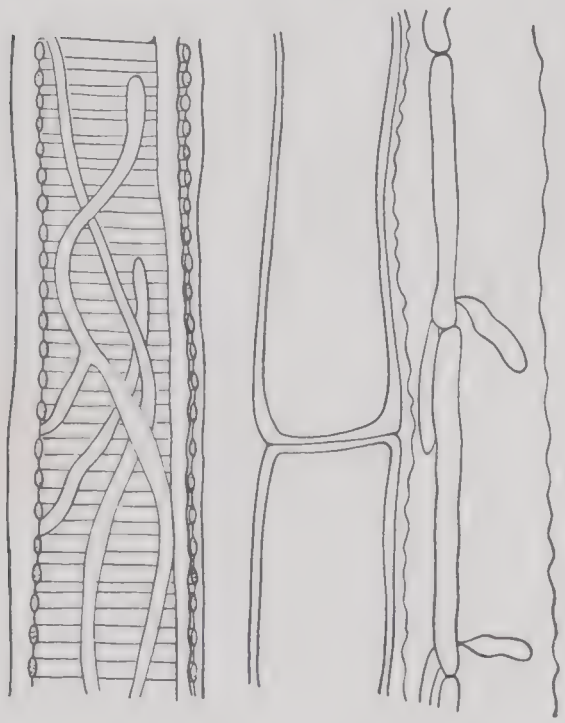

FIG. 66. The cabbage-yellows organism in tracheae of the cabbage plant. Note formation of microconidia in the vessel. (*After Gilman.*)

Varietal Resistance. Since the only successful control of yellows is through the development of resistant varieties, the nature of resistance to this pathogen has received much study. Jones and Gilman (8) noted in 1909 that in heavily infested fields in Racine County, Wisconsin, occasional plants survived and showed no signs of disease throughout the season. They selected a number of such individuals and grew them to seed the following season, allowing the plants to cross-pollinate, since cabbage is highly self-incompatible. When plant-to-row trials were made

in the next generation on infested soil, the progenies were much higher in percentage of surviving plants. Continued selection for two more generations led to a highly resistant stock, which was introduced in 1916 as a new variety, Wisconsin Hollander. This continues to be used widely in this country. By similar procedures, Jones *et al.* (9) developed two other varieties, Wisconsin All Seasons and Wisconsin Brunswick, which were released in 1920. These three varieties were not completely resistant and in extremely warm seasons large percentages of plants showed traces of yellows during the warmest part of the summer and then recovered to produce almost normally. Wisconsin Brunswick is no longer grown, but Wisconsin All Seasons is used widely.

Walker (15) developed inbred lines by selfing plants by bud pollination. He secured some lines which were completely free from yellows under favorable conditions for the disease. In such lines it was shown that resistance was controlled by a single dominant gene (15). Anderson (1) studied Wisconsin Hollander by producing inbred lines and found that resistance in that variety was inherited as a multiple-gene quantitative character. This established the fact that two types of resistance to yellows occurred. One was inherited qualitatively (type A) and the other quantitatively (type B). Blank (5) showed that both types occurred in Wisconsin All Seasons. Walker and Smith (20) showed that the two types could be distinguished by growing progenies in infested soil at a constant temperature of 24°C., at which temperature only type A individuals survived. By use of this technique much more rapid progress could be made than by field selection. Since 1928 nine type A resistant varieties representing a range of horticultural type have been developed. They are described in U.S. Department of Agriculture Farmers' Bulletin 1439 (17) and in several other publications (16, 18, 21, 23, 24). Blank (4) studied collections of the fungus from several regions of the United States and found no variation in selective pathogenicity.

Smith and Walker (11) and Anderson and Walker (2) studied the relation of the pathogen to susceptible, type B resistant, and type A resistant plants. In the susceptible plants penetration of the young root tissue occurred, and the fungus became established in the vascular system promptly. In type B resistant plants penetration occurred in a similar manner, but less abundantly, and the fungus was less aggressive in the vascular system. In type A plants the fungus penetrated the cortex of young roots but did not progress as far as the spiral vessels. Although no cytological or histological reaction of the resistant host was clearly defined, it was evident that the young root tissue of type A resistant plants was incompatible with the fungus and the latter was effectively excluded from the vascular system.

Walker and Hooker (22) studied susceptible, type B resistant, and type A resistant plants in sand nutrient culture in which the concentration and balance of the nutrient was varied. Type A plants were not affected as to resistance by variation of nutrient. In type B resistant plants the amount of disease was increased as concentration was decreased, just as in susceptible plants, although the disease was always less severe in the former than in the latter.

The disease likewise increased in type B resistant plants when low-potassium nutrient was supplied, as was the case in susceptible plants. Quantitative resistance is therefore influenced much more by temperature and nutrition than is qualitative resistance.

Control. The yellows disease is successfully controlled by the use of resistant varieties. A list of the available recommended resistant varieties and their general characteristics follows:

Variety	Type of Resistance	Season	Head shape
Jersey Queen	A	Early	Pointed
Wisconsin Golden Acre	A	Early	Spherical
Resistant Detroit	A	Early	Spherical
Racine Market	A	Second early	Spherical
Marion Market	A	Midseason	Spherical
Globe	A	Midseason	Spherical
All Head Select	A	Midseason	Flat
Improved Wisconsin All Seasons	A	Late	Flat
Improved Wisconsin Ballhead	A	Late	Spherical
Resistant Flat Dutch	B	Late	Flat
Wisconsin Hollander	B	Late	Semispherical
Bugner	B	Late	Semispherical
Resistant Red Hollander	A	Late	Spherical

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FUSARIUM WILT OF TOMATO

In referring to the wilt of tomato incited by *Fusarium oxysporum* f. *lycopersici*, it is necessary to use a qualifying term (*Fusarium* wilt) because there are two other important wilt diseases of this host, *i.e.*, *Verticillium* wilt (*Verticillium albo-atrum* Reinke & Berth.) and bacterial wilt (*Pseudomonas solanacearum* E. F. Sm.). *Fusarium* wilt was first noted in the Channel Islands some few years before 1895. It occurs in northwestern Europe on tomatoes under glass. It is known in many of the warm regions throughout the world. In the United States it is found in

the latitude of Wisconsin only under glass, but in regions to the south of this latitude it is an important field disease. The pathogen is restricted to common tomato, *Lycopersicon esculentum* Mill., and the red-currant tomato, *Lycopersicon pimpinellifolium* Mill.

Symptoms. In young plants in the greenhouse, two of the earliest symptoms are clearing of the veinlets (12) and drooping of the petioles (31). In the field the disease may appear at any time that conditions are favorable. Yellowing of the lower leaves appears first, usually affecting the leaflets unilaterally. The affected leaves die, and the symptoms continue to appear on successively younger leaves. One or more branches may be affected while others remain symptomless. After the disease has advanced for a few weeks, browning of the vascular system may be seen in cross sections of the lower stem. The plant as a whole is stunted, and commonly it eventually goes into a permanent wilt of the leaves, which die as they cling to the upright woody stems.

The Causal Organism. *Fusarium oxysporum* f. *lycopersici* (Sacc.) Snyder & Hansen, 1940.

Synonymy:

Fusarium oxysporum Schl. subsp. *lycopersici* Sacc., 1886.
Fusarium lycopersici Sacc.
Fusarium bulbigenum (Cke. & Mass.) *lycopersici* (Brushi) Wr. & Reinking, 1935.

The morphology and cultural characteristics are similar to those of the cabbage-yellows organism. Like most fusaria this organism grows well on many media and tolerates a wide range of pH in the substrate. It has an optimum temperature for growth at about 28°C. A number of investigators have shown that isolates vary considerably in their growth reactions and in pathogenicity (16, 17, 32 37). Alexander and Tucker (2) have described a race of the fungus, thus far rarely found in nature, which is pathogenic on strains of the host highly resistant to other isolates.

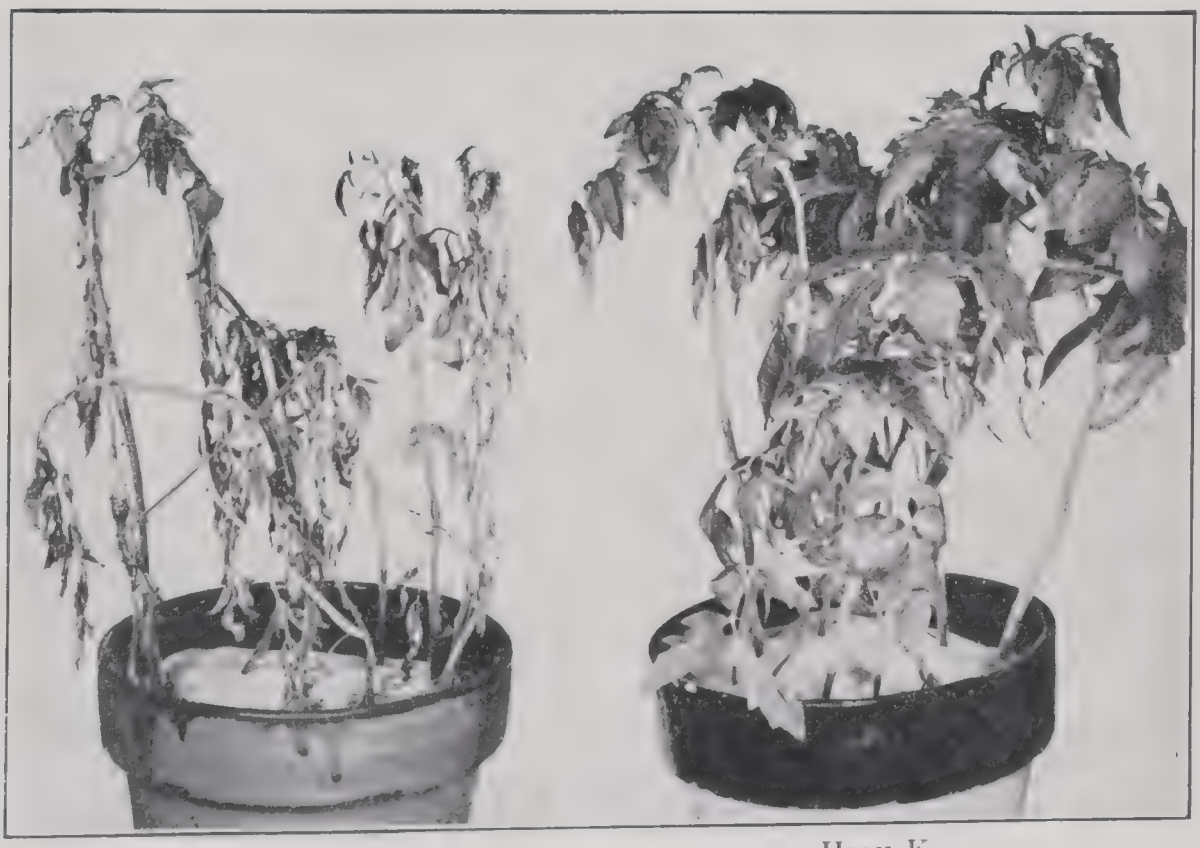
Disease Cycle. The fungus becomes established readily in many soil types, such soils remaining infected almost indefinitely. Elliott and Crawford (9) in Arkansas described transmission of the pathogen with the seed, as did Kendrick (18) in California. Samson *et al.* (24), in Indiana, found it to occur only rarely in seed extracted from infected plants. The chief method of wide distribution is by transplants, while local dissemination is by transplants, wind-borne soil, surface drainage water, water-borne soil, and implements.

The range of the disease indicates that it is restricted by cool climate more than cabbage yellows is. Clayton (5, 6) studied soil moisture and

air and soil-temperature relations. He found that the optimum soil temperature is 28°C. The disease was inhibited above 33° and below 21°. Air temperature had a similar effect. Soil moisture conducive to best vegetative growth of the host was most favorable to disease development. Sherwood (27) found that the pathogen grew well in a range of pH in the culture medium from 3.6 to 8.4. Scott (25, 26) found two maxima in the growth curve, one at pH 4.5 to 5.3, the second at 5.85 to 6.85; for spore germination the two maxima were near 4.5 and 7.0, respectively. He suggested that the living mycelium behaves like an amphoteric colloid with an isoelectric point at or near pH 5.4. Sherwood (27) found that in tomato seedlings the percentage of wilted plants decreased as the pH of the infected soil increased up to about 7.4. Scott (25, 26) found maxima for wilt production below 6.4 and above 7.0. Foster and Walker (13) showed that the conditions under which the host plants were grown before infection had a bearing upon the rate and degree of disease development. Air and soil temperatures conducive to best growth of the host produced plants which succumbed more severely than when plants were predisposed at higher or lower temperatures. Other conditions which predisposed plants to wilt were low soil moisture; short day length; low light intensity; nutrients low in nitrogen, high in potassium, low in phosphorus, or low in pH. Walker and Foster (29) showed that the rate of disease development decreased with increase in concentration of a balanced nutrient. Low-potassium and high-nitrogen nutrients increased disease development while high potassium and low nitrogen had an opposite effect (Fig. 67).

The nature of the action of the pathogen in inducing wilt has received much study. Mechanical plugging of the vascular system by the fungus has been suggested, but this has been discarded by most authorities in favor of the toxicity theory. The latter suggests that by-products of the pathogen produced in the vessels are transported throughout the plant to cause the various pathological symptoms. Many have shown that the filtrate from liquid cultures of the fungus contains one or more substances which cause wilting of shoots when the cut stems are inserted into the liquid. Such a response can be secured, however, with the filtrate from many organisms nonpathogenic to tomato. Gäumann *et al.* (4, 14) have designated a polypeptide, named lycomarasmin, produced by the wilt organism as one of the chief toxic materials which is responsible for wilt. They postulated that as this material enters the host cells from the transpiration stream, the selective permeability of the plasma membrane is altered. The excessive loss of water which follows initiates disease development, and the extent depends upon the amount of damage by the toxic material.

Low N High N



Low K High K

Fig. 67. The effect of mineral nutrients on *Fusarium* wilt of tomato. The rate of disease development is enhanced when the nutrient is high in nitrogen or low in potassium.

Varietal Resistance. The first work on the development of resistant varieties was by Essary in Tennessee, which he reported in 1912 (10). Edgerton in Louisiana was next to carry on breeding for wilt resistance, introducing the varieties Louisiana Pink and Louisiana Red. Pritchard (22) of the U. S. Department of Agriculture headed up the most extensive program in this country, which produced a number of successful resistant varieties, including Marglobe, Norton, and Pritchard (19). Various other varieties for field and greenhouse use have been developed in a number of states. A list of recommended varieties is given by Doolittle (8).

Resistance in the varieties mentioned above was not completely fixed and behaved as a quantitative genetic character comparable to type B yellows resistance in cabbage. Bohn and Tucker (3) announced a high type of immunity to wilt which is comparable to type A yellows resistance in cabbage and is controlled by a single dominant gene. This was found in a strain of red-currant tomato. Walker and Foster (29), studying nutrition in relation to disease development, found that while the resistance of Marglobe was markedly influenced by the type and concentration of host nutrition, the high resistance of red-currant tomato was not affected. The first commercial tomato with type A resistance to *Fusarium* wilt was introduced in 1941 as Pan America (20, 21).

Control. The only practical means of control of *Fusarium* wilt is by the use of resistant varieties of tomato (8).

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EARLY BLIGHT OF POTATO AND TOMATO

One of the most common foliage diseases of potato and tomato is that known as early blight. Its range is almost coexistent with the two crops, and it is much less limited by climatic conditions than late blight. While the pathogen incites a tuber rot of potato and a fruit rot of tomato, its greatest effect is due to injury to the foliage as a leaf spot and as an instigator of premature defoliation. The pathogen was first described by Ellis and Martin in 1882 from dying leaves of potato collected in New Jersey. Distinction of the disease from other foliage troubles began about 1891. The most critical early work was that of Jones (11–15) at Vermont during the period from about 1891 to 1903. Rands (25) was the first to prove that the same pathogen incited the leaf and stem disease on both potato and tomato. Tuber rot was overlooked until 1925, when it was described in Maine by Folsom and Bonde (6).

Prior to about 1945 its damage on potato was difficult to estimate because of the usual coexistence with leaf-hopper damage. Now that improved insecticides have made it possible to control the leaf hopper almost completely, the true effect of early blight can be determined more accurately. There is little doubt that it remains an important disease both on potato and tomato and one which is among the most difficult to control with fungicides.

Besides potato and tomato the following are known to be hosts of the pathogen: *Solanum aviculare* Forst., *S. carolinensis* L., *S. commersoni* Dun., *S. giganteum* Jacq., *S. melongena* L., *S. nigrum* L., *S. nigrum guinense* L., *S. rostratum* Dun., *S. warscewiczii* Hort., *Hyoscyamus niger* L., *Hyoscyamus albus* L., *Lycopersicon pimpinellifolium* Mill., and *Nicandra physaloides* Gaertn.

Symptoms. The disease appears first as spots on the leaflets of tomato and potato. These are dark brown to black, and in the leathery-appearing necrotic tissue concentric ridges often form to produce a characteristic target-board effect. The spots are oval or angular in shape and on leaflets are mostly up to 3 or 4 mm. in diameter. There is usually a narrow chlorotic zone around the spot, which fades into the normal green. While

the spots are quite delimited, there is ample evidence of pathological effects beyond the necrotic tissue. When spots are numerous, the general effect on the leaflet is such as to make it go into a senescent condition. Lowest leaves are affected first, as a rule, and the disease progresses upward. The leaves of potato dry up and droop while in the tomato they are more inclined to drop off (Fig. 68).

FIG. 68. Early blight of potato on tuber and leaf.

Spots may develop on potato stems, but they are much less common and destructive than on tomato. In the latter plant dark spots, often target-boarded, occur anywhere on the stem. They are especially injurious when they occur at the juncture of stem and side branch, weakening the latter so that it breaks when the growing fruits provide sufficient weight. Collar rot on tomato is a form of stem lesion which begins in the seedling stage. Lesions form at the soil line, extending above and below that point to form cankers or to girdle the stem, resulting in worthless plants (Fig. 69).

On potato tubers the surface lesions are a little darker than the healthy

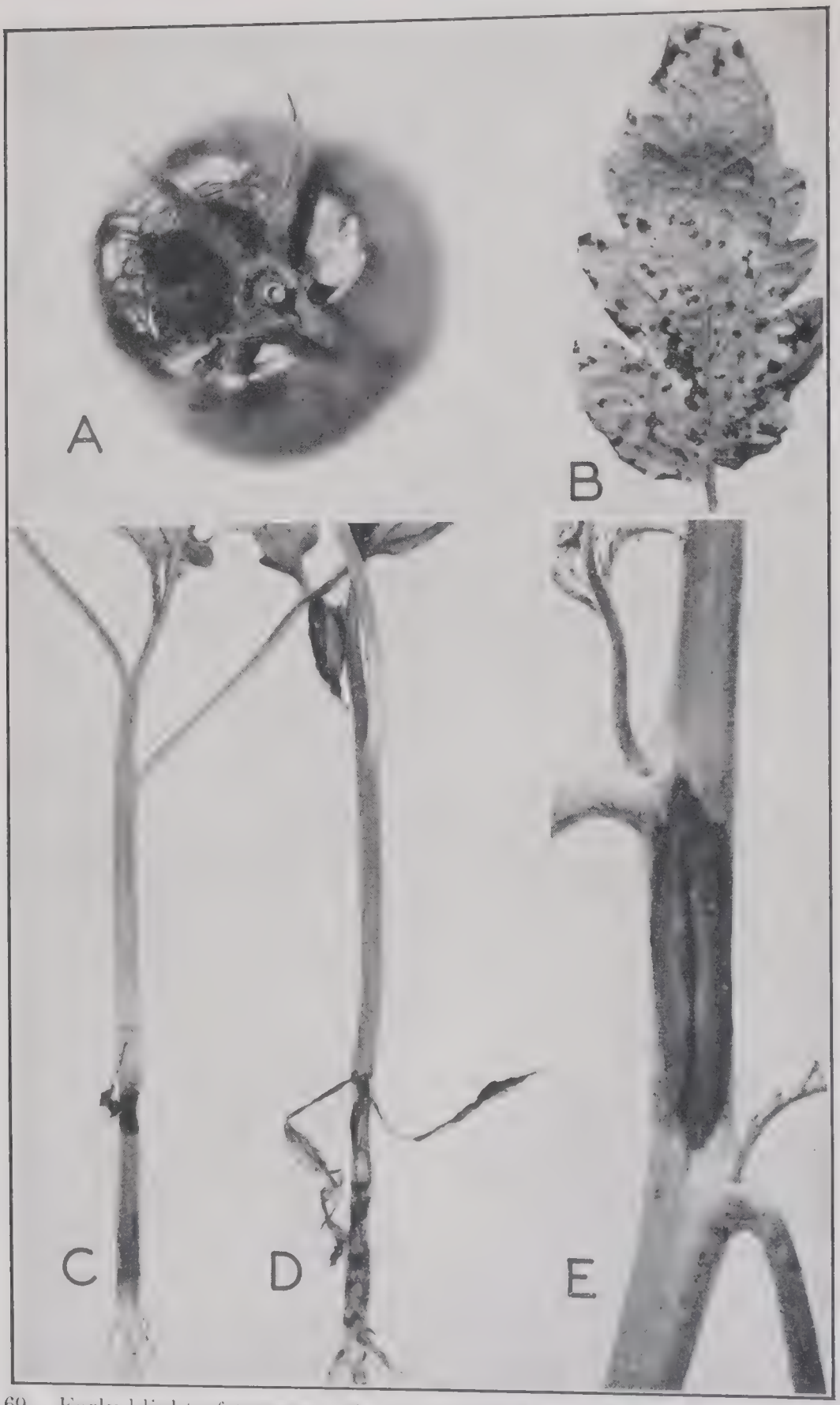

FIG. 69. Early blight of tomato. *A*, fruit rot progressing from sepal infection; *B*, leaf spot; *C*, collar rot starting from an infected, drooping cotyledon; *D*, advanced collar rot; *E*, zonate stem lesion.

skin. They are slightly sunken, circular or irregular in shape, and vary in size up to 2 cm. in diameter (Fig. 68). The margin between healthy and diseased tissue is usually quite well defined and is sometimes slightly raised. The underlying tissue shows a brown, corky dry rot usually not more than 6 mm. in depth. Fissures may develop in older lesions.

Tomato fruits are infected in the green or ripe stage, usually at the stem end but also through growth cracks and other wounds. Lesions start as black or brown, usually sunken, spots which may enlarge to the extent that they involve most of the fruit (Fig. 69). The diseased tissue tends to appear leathery in consistency and may bear a velvety black mass of spores over the surface. Sometimes a small lesion at the surface is only part of an extensive internal region of decayed tissue.

The Causal Organism. *Alternaria solani* (Ell. & G. Martin) L. R. Jones & Grout, 1896.

Synonymy:

Macrosporium solani Ell. & Mart., 1882.
Macrosporium solani Cke. (in part), 1883.
Macrosporium cookei Sacc. (in part), 1896.
Alternaria solani Sorauer (in part), 1896.
Sporidesmium solani var. *varians* Vanha, 1904.
Alternaria porri (Ell.) Neerg. f. sp. *solani* (Ell. & G. Martin) Neerg., 1945.
Alternaria dauci (Kühn) Groves & Skolko f. sp. *solani* (Ell. & G. Martin) Neerg., 1945.

The mycelium is septate and branched and becomes dark-colored with age. Conidiophores, rising from the older diseased host tissue, are relatively short and dark-colored. Conidia (12 to 20 by 120 to 296 μ) are beaked, muriform, dark-colored, borne singly or in chains of two (in pure culture). They form from a bud which arises from the terminal cell of the conidiophore (Fig. 70). The fungus grows readily on artificial media, usually producing an abundance of yellowish to reddish pigment which diffuses through the substrate. It fruits very sparsely in pure culture, as a rule, but abundant sporulation is stimulated by wounding the mycelium or by exposing the culture to ultraviolet rays (18, 24). Considerable variation between isolates has been observed with reference to virulence and to growth and sporulation in pure culture (4).

Disease Cycle. The mycelium remains viable in dry infected leaves for a year or more, and the conidia remain viable for as long as 17 months at room temperature. In the extraction of tomato seed from infected fruits contamination of the seed may occur readily. Overwintering may take place in infected plant debris and on tomato seed and in potato tubers. Conidia germinate within 1 to 2 hr. at temperatures from 6 to 34°C., and

in 35 to 45 min. at the optimum, 28 to 30°. The cardinal temperatures for growth in pure culture are minimum, 1 to 2°; optimum, 26 to 28°; maximum, 37 to 45° (23, 25). The fungus penetrates leaf and stem tissue directly through the epidermis (Fig. 187). Under conditions of favorable temperature and moisture, spots become visible within 2 to 3 days, and spores may be produced within the next 3 or 4 days. Spore production

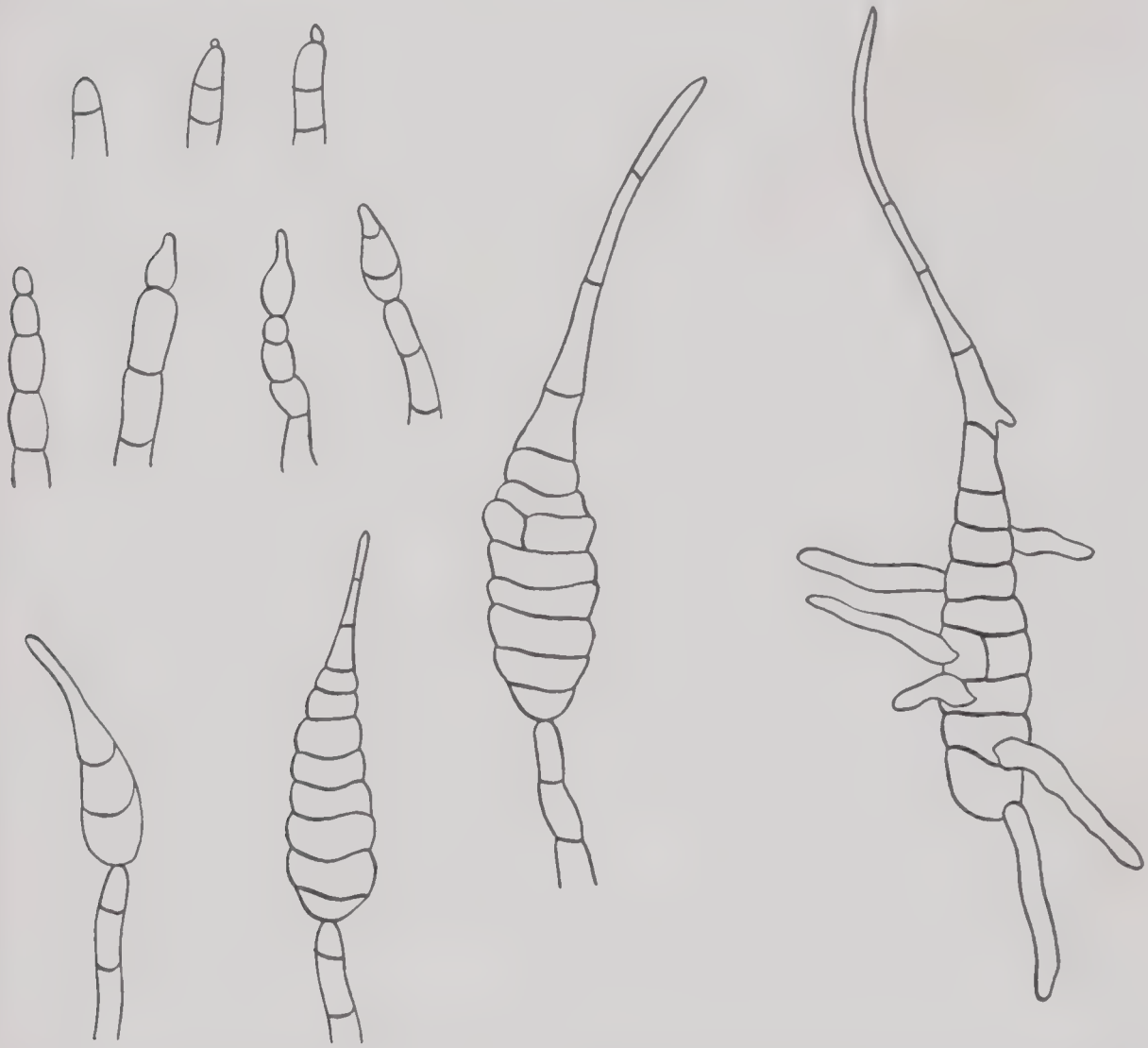

FIG. 70. Stages in the development and germination of the conidium of *Alternaria solani*. (*After Rands.*)

usually begins when the leaf spot is about 3 mm. in diameter. Heavy dews with frequent rains seem to be essential for abundant sporulation (25). The conidia are readily detached and are disseminated chiefly by air currents. Colorado beetles (25) and flea beetles (9) facilitate infection by producing wounds, and they may possibly carry spores on their bodies.

On tomato seedlings infection occurs on the cotyledon when an infected

seed coat remains attached to the latter after emergence. Such a cotyledon usually dies and droops down without becoming detached, so that it lies in contact with the hypocotyl. The fungus then enters the hypocotyl; it may also enter directly from infected debris in the soil. Collar-rot lesions begin in these ways. Secondary spread to stems and foliage follows. In potato, primary infection usually appears on the foliage.

It is characteristic of this disease that its appearance as a foliage blight on potato or tomato usually coincides with the time when the plants have begun formation of tubers or fruits. While much still remains to be learned about the epidemiology of the disease, it is commonly observed that an important factor is predisposition of the host by climatic or soil conditions which tend to reduce its physiological vigor. Conversely, high soil fertility tends to reduce the severity of the early blight, especially in tomato (27). Older leaves are usually affected first, and younger leaves become affected in turn as they reach a certain degree of physiological maturity. When tomatoes are fruiting heavily, and when potatoes are setting tubers, the entire plants appear to become more susceptible, and this is thought by some to be due to the physiological stress placed upon the photosynthetic region of the plant by heavy translocation to developing storage organs.

Gratz and Bonde (7) found 13 to 16°C. to be the optimum for tuber rot of potato with little development at 5 to 7° and at 25°. Nightingale and Ramsey (23) found that decay was more rapid in ripe than in green tomatoes and that the temperature for optimum rate of decay (26 to 28°) coincided with that given for growth in pure culture by Rands (25). The temperature relations of foliage blight have not been worked out critically, but with at least some strains of the pathogen cool temperatures are most favorable (27). In fact, the disease is usually more important in the northern part of the tomato canning belt extending from New Jersey to Missouri than in the central latitude of this region. However it is a relatively rare disease in the British Isles.

Control. Crop rotation is an important control measure, but because of the wind-borne nature of the conidia it can be depended upon only to delay the initial appearance of the disease. In so far as foliage blight is concerned, varietal resistance is of little value although there are some differences in tolerance of both potato and tomato varieties. Andrus *et al.* (1) have reported a high degree of resistance to collar rot in certain exotic varieties of tomato. The inheritance of the resistance is controlled by a single major gene. The variety Southland was the first developed for American use which contains collar-rot resistance.

Disinfection of seed is essential and is accomplished by treatment with New Improved Ceresan, applied either as a soak for 5 min. in 1–1,200

concentration or as a dust at the rate of 0.5 per cent of the weight of the seed.

The use of fungicides to control early blight has received much attention in tomato and potato, usually in conjunction with control of late blight, hopper burn, and chewing insects. By and large, early blight is not so readily controlled as late blight, especially with Bordeaux mixture. Insoluble coppers are only moderately effective. Greater success is attained with zinc carbamates, and of these Zerlate and Parzate are the most effective. Since Zerlate is not reliably effective against late blight, an alternating schedule of Zerlate and an insoluble copper has been suggested for tomato. Certain adjuvants improve the effectiveness of Zerlate (19). It is not possible to give an over-all recommendation for either potato or tomato, and therefore local experiments and recommendations should be followed.

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ONION SMUDGE

This disease was first described by Berkeley in England in 1851. It is common in Europe and throughout northeastern and midwestern United States. It occurs principally upon white-bulb varieties, and its importance consists chiefly in the reduction in market value due to blemish of the outer scales. Slight shrinkage and premature sprouting occur in storage. The disease affects shallot (*Allium ascalonicum* L.) and leek (*A. porrum* L.).

Symptoms. Smudge is confined almost entirely to the scales and lower portions of the unthickened leaves. It first becomes manifest upon the appearance of minute stromata which form just beneath the cuticle of the host; they are dark green at first, becoming black with age. The stromata may be scattered promiscuously over the surface of the bulb, or they may be congregated in smudgy spots around a few centers of infection (Fig. 71). The spots are roughly circular and occasionally contain stromata arranged in concentric rings. Under moist conditions the stromata bear acervuli which contain prominent black setae readily distinguished with a lens of low magnification. Cream-colored spore masses frequently form on these fruiting bodies. On underlying scales similar spots are surrounded by yellowish borders. On the fleshy scales the disease appears underneath spots on outer scales or where the fleshy scale is exposed to the soil (Fig. 72). It appears as minute sunken yellowish spots, which gradually enlarge and often coalesce. The black stromata may appear as the scale

dries down, resulting in spots not unlike those on the dry outer scales. If the fleshy scale dries down before stromata appear, slightly raised yellowish spots result, giving to white onions an unnatural color which is almost as detrimental to their market value as the black, smudgy spots.

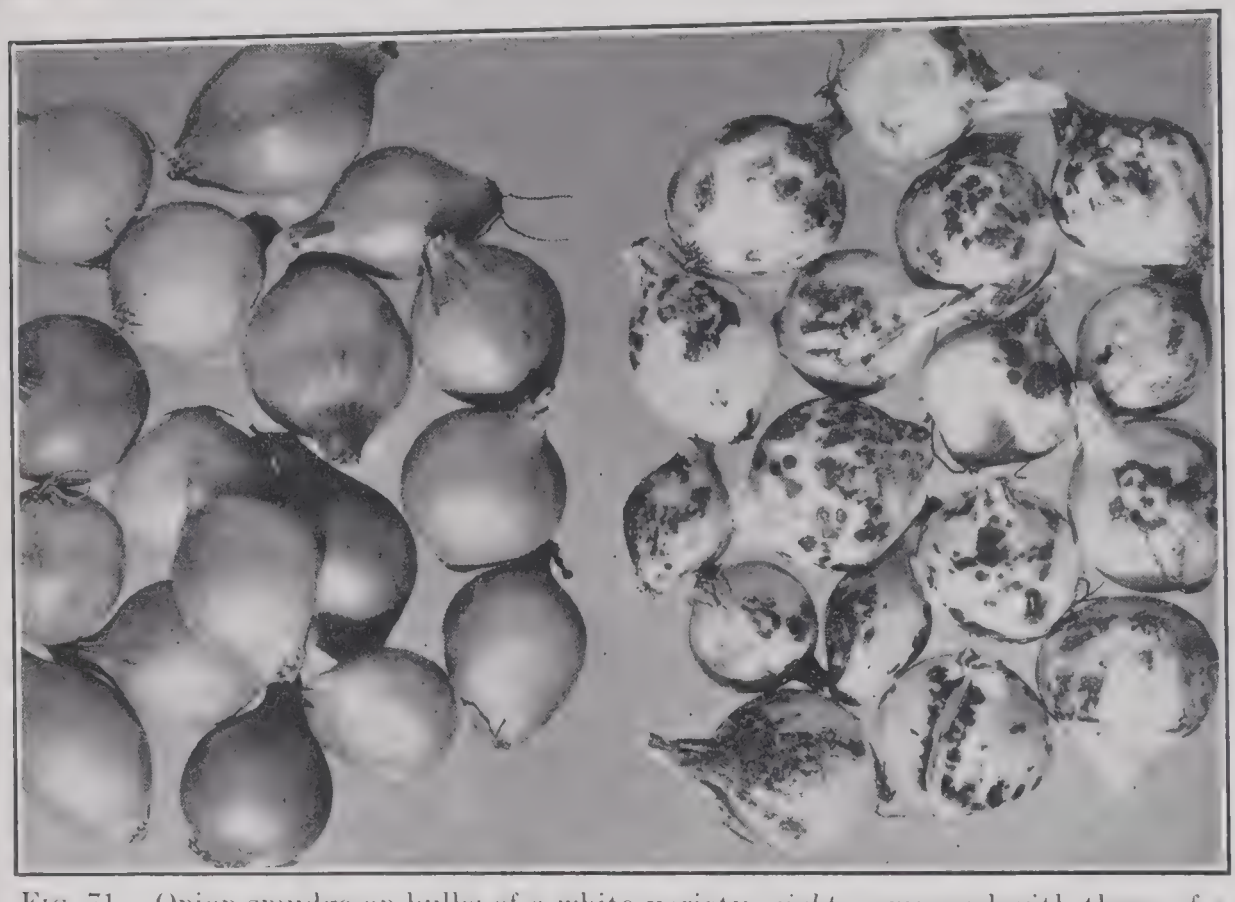

FIG. 71. Onion smudge on bulbs of a white variety, *right*, compared with those of a resistant yellow variety, *left*, grown under the same conditions in infested soil.

The Causal Organism. *Colletotrichum circinans* (Berk.) Vogl., 1907.

SYNONYMY:

Vermicularia circinans Berk., 1851.
Volutella circinans (Berk.) Stevens & True, 1919.
Cleistothecopsis circinans (Berk.) Stevens & True, 1919.

The mycelium is septate, branching, hyaline when young, the walls becoming darker, and thicker and the cytoplasm denser with age. Intercalary chlamydospores are formed. By close intertwining of thick-walled hyphae, dark green to black stromata, a fraction of a millimeter in diameter and a few to several hundred microns thick, are formed, sometimes coalescing or being connected by dark-walled mycelium. Acervuli are formed on stromata beneath the cuticle by formation of a palisade layer of short, hyaline conidiophores which ruptures the cuticle (Fig. 73). Scattered through the acervulus are numerous thick-walled, dark-colored setae,

up to three-septate, upwardly attenuate, 80 to 315 μ long, and arising from the basal stroma. Conidia are borne acrogenously, being budded off at the tip of the conidiophore one at a time. They are fusiform, nonseptate, hyaline to slightly ocherous, somewhat curved and obtuse at the apex, 3 to 6 by 14 to 30 μ, mostly 3 to 4 by 18 to 28 μ. Under favorable conditions they accumulate in cream-colored gelatinous masses. They germinate by one, occasionally two or three, germ tubes (Fig. 74). Appressoria are formed abundantly when the germ tubes come in contact with the host surface; they are thick- and dark-walled and measure 4 to 5 by 6.5 to 8μ. From the appressorium an infection tube is formed which penetrates the host cuticle. Stevens and True described an ascigerous form on onion scales affected with smudge which they assumed to be the perfect state of *C. circinans*, but this observation has never been confirmed.

FIG. 72. Smudge on bulb of a yellow resistant variety, the outer scale of which was broken, thereby exposing the fleshy scale to the organism in the soil. (*After Hatfield, Walker, and Owen.*)

Disease Cycle. The fungus probably persists in the soil as stromata, or as a free-living saprophyte, for several years in the absence of the host. Conidia are quite sensitive to freezing and drying but, when in desiccated spore masses, withstand several weeks of unfavorable conditions. Good germination occurs from 13 to 25°C. with an optimum at 20°; best growth on agar occurs at about 26°. The disease develops from about 10 to 32° with an optimum about 26°. Moist weather is essential for production of conidia, and spattering rain is necessary for dissemination.

The invasion of the host is accomplished by germination of the conidium and formation of an appressorium, which, by means of a gelatinous exterior, becomes fastened to the cuticle. An infection peg penetrates the cuticle by mechanical pressure, and the mycelium then develops, first between the cuticle and the outer epidermal wall (Fig. 74). By enzymic action the cellulose wall is softened; it thickens and becomes striate, while the mycelium penetrates its layers and subsists on it (Fig. 185). By this time the protoplast of the underlying epidermal cell shows signs of change in permeability and in general appearance, indicating signs of degeneration. Mycelium penetrates the epidermal cell lumen but probably not

until the protoplast has been affected in advance. The fungus continues to invade the tissue, progressively softening cell walls, penetrating the walls and the cell lamina. Johnson (4) showed that the fungus penetrated

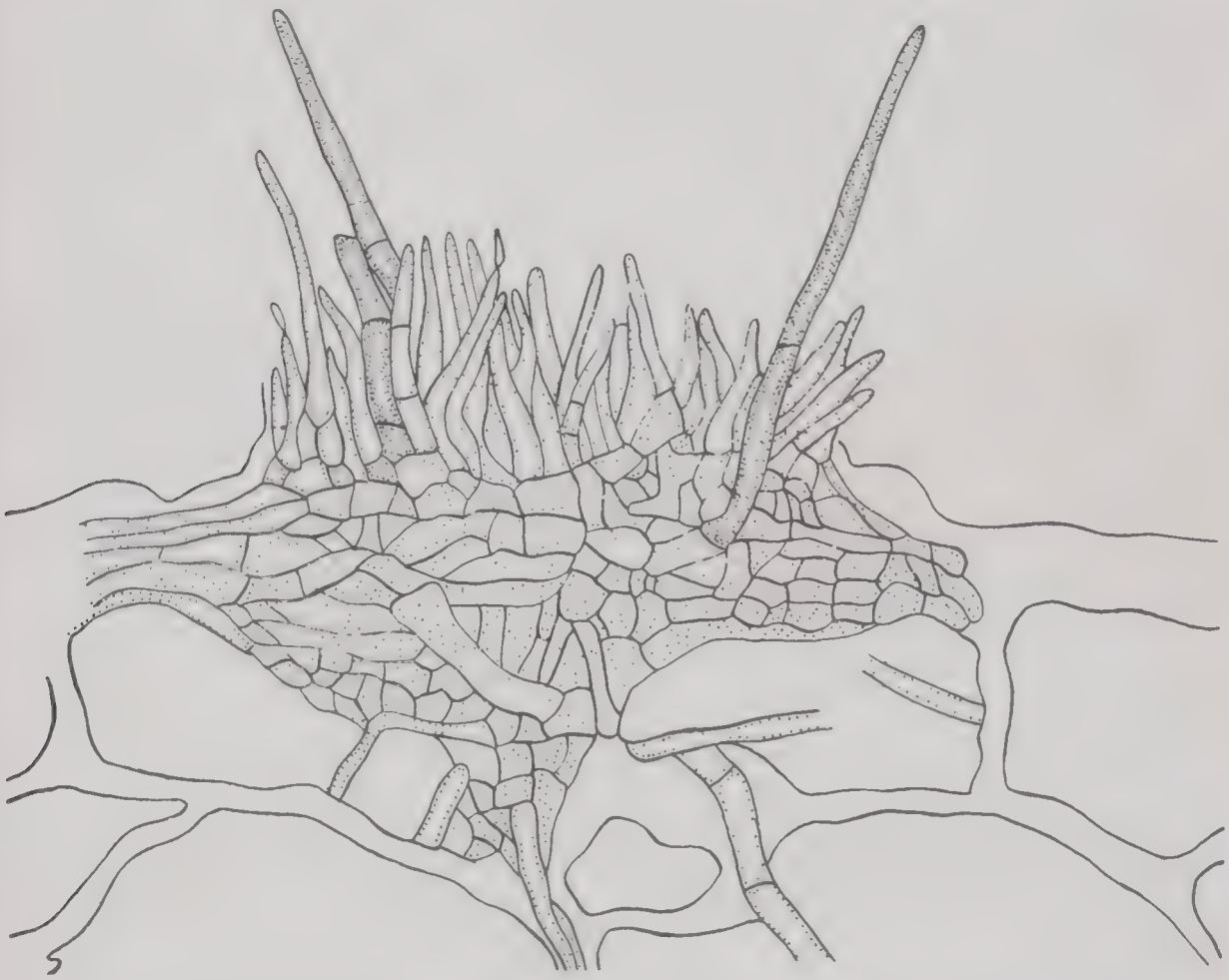

FIG. 73. Acervulus of *Colletotrichum circinans* rising from the stroma, which developed in the epidermal wall. Note rupture of the cuticle of the host.

in a similar way plants of some 20 other species, and in some of these lesions were produced.

Varietal Resistance. In the first description of the disease in 1851 Berkeley stated that it occurred on white onions while colored varieties growing alongside were free (Fig. 71). This has been observed many times since, and the disease has received much study from the standpoint of the nature of disease resistance. Walker (11, 12) emphasized the close association of the red or yellow pigment in the dry outer scales with resistance to the pathogen. Even in the same scale, adjoining colored and colorless tissues were resistant and susceptible, respectively. A water-soluble material in colored scales was shown to prevent germination of conidia, cause the rupture of young germ tubes, or permit only abnormal germ tubes without formation of the essential appressoria (Fig. 75). This toxic ma-

terial also affected the spores of *Botrytis allii*, the incitant of neck rot, in which a similar correlation of colored varieties and resistance is known. The material had little effect on spores of the black-mold fungus (*Aspergillus niger* v. Tiegh.), which affects colored varieties as readily as white varieties. From extracts of colored outer scales two colorless water-soluble phenolic compounds were isolated which accounted for a considerable proportion of the toxicity of the water extracts (1, 6–8). These are protocatechuic acid and catechol. It was shown that these materials function

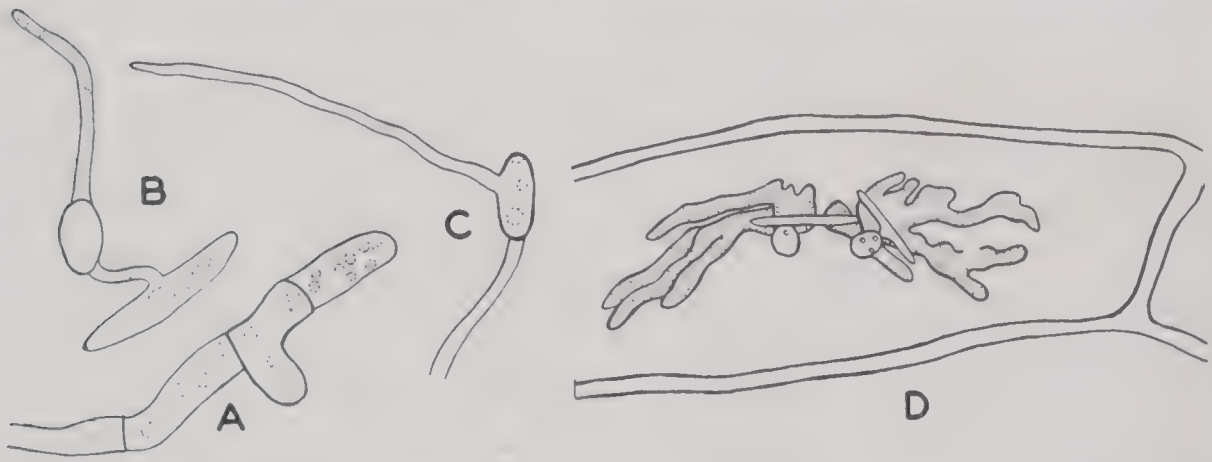

FIG. 74. *Colletotrichum circinans*. *A*, chlamydospore; *B*, conidium which has germinated and formed an appressorium, which in turn has germinated; *C*, germinating appressorium; *D*, conidia and appressoria on the surface of the host and the subcuticular mycelium developing after penetration.

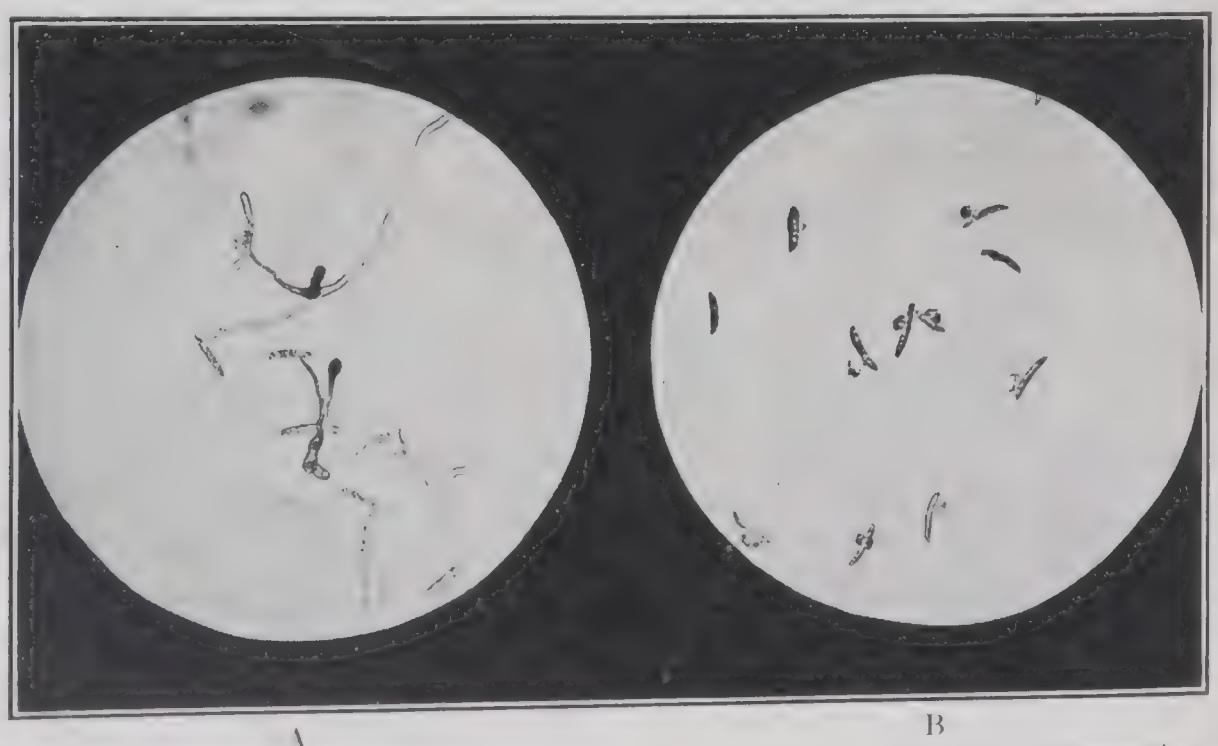

FIG. 75. *Colletotrichum circinans*. *A*, normal germination of conidia and formation of appressoria in water extract of dry outer scale of white susceptible variety; *B*, ruptured germ tubes from conidia in water extract of dry outer scale of colored resistant variety containing soluble protocatechuic acid and catechol.

by diffusing out of the dry colored scales to drops of water on the surface which contain spores of the pathogen and preventing or inactivating germination before infection takes place.

It was shown by Walker (12) that if the dry outer colored scales were removed and drops of water containing spores were placed on the uninjured surface of the fleshy colored scale, infection took place and the disease progressed as rapidly as in fleshy white scales. In the case of colored fleshy scales normal penetration occurred; the fungus developed in the gelatinized epidermal wall. Before it penetrated the cell lumen the pigment, which

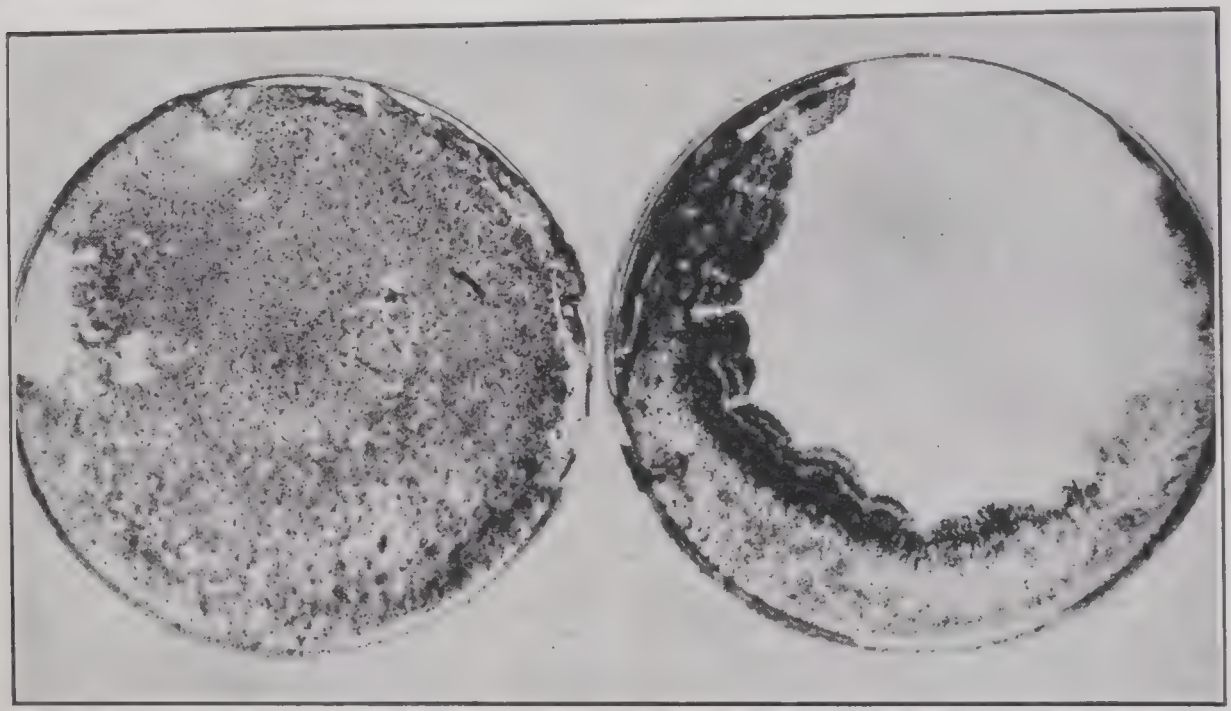

FIG. 76. Agar plates seeded with conidia of *Colletotrichum circinans* and incubated at 20°C. Normal growth ensued in the plate at left. In the plate at right a drop of extract of fleshy onion scale was placed inside the dish, not in contact with the agar. The volatile toxic materials released upon crushing of the onion tissue killed the conidia except around the outer edge of the agar plate.

is normally confined to the outer epidermal cells of fleshy scales, disappeared. In a normal fleshy scale the diffusion of cell solutes through the epidermal wall and cuticle is very slight, while it occurs readily in dry outer scales, since no plasma membrane interferes. Consequently the relation of host cell to pathogen in the dry outer scales is quite different from that in the fleshy scales.

Walker and associates (3, 9, 11–14) showed that there was another type of antibiotic in the fleshy scales of white and colored varieties which was very toxic to the conidia and mycelium of *C. circinans* and of certain other fungi. This is in part volatile (Fig. 76). Why the smudge pathogen penetrates and parasitizes tissue which contains material so toxic to it is not understood, but it may be that the same action of the pathogen on

the cell in advance of cell penetration is a factor in detoxification before invasion. It has been shown that the neck-rot fungus is less sensitive to the fleshy-scale antibiotics than is the smudge organism, and the fact that this is so may be one reason why the former causes a quite rapid decay while the latter produces a very slow decay of onion bulbs (3, 9, 14). On the other hand strongly pungent varieties are more resistant to neck-rot than are mildly pungent varieties (3, 9).

Turning again to outer-scale resistance associated with water-soluble phenolic substances, it may be expected that weather conditions might influence their effectiveness, *e.g.*, continuous rain might dilute the phenols and decrease resistance. Hatfield *et al.* (3) studied the same colored and white varieties over a period of 4 years, and when the amount of disease was converted to an index figure for each, it was found that the index varied for each variety from year to year. While the same general relation of color to resistance was maintained, the index was generally higher when conditions were very favorable for the disease. Moreover, the indices for certain colored varieties sometimes were as high as those for white varieties. This was shown, in the main, to be the case for colored varieties which had relatively few outer protective scales and in which the scales tended to slough readily and expose fleshy scales to infection before harvest. Owen *et al.* (9) showed that the volatile substances in strongly pungent varieties were supplementary factors in reducing the amount of disease as compared with that in mildly pungent varieties.

The inheritance of outer-scale resistance is, so far as present evidence goes, identical with the inheritance of color, and it would appear that the same genes which control the metabolism of the phenolic color compounds, also control the metabolism of the simpler, water-soluble phenolic substances which inactivate the pathogen. The genetics of color in onion has been worked out by Rieman (10) and by Clarke *et al.* (2). Three pairs of genes are involved. The dominant gene *C* is necessary for the production of red or yellow pigment. The dominant gene *R* is responsible for the production of red color, if *C* is present. The gene *r*, recessive to *R*, is responsible for the production of yellow pigment in the presence of *C*. If *c*, the allele of *C*, is homozygous, the bulbs are white even though *R* or *r* is present. In the third factor pair, the gene *I* is partially dominant over its allele *i*. *II* completely inhibits color even though *R* or *r* is present along with *C*. *Ii* in the presence of *R* and *C* results in a dilute red color and in the presence of *r* and *C*, in a light yellow or cream color.

The inheritance of bulb color is of interest here because the genes which control the metabolism producing color also control the metabolism of the antibiotics responsible for disease resistance. White bulbs may have the gene for red or the one for yellow, but the metabolism for color and

for resistance does not operate if *I* is present in the homozygous condition or if *c* is present in the homozygous condition. Thus the following genotypes and phenotypes may occur:

Genotype	Phenotype	Resistance
RR CC ii	Red	High
Rr CC ii	Red	High
RR Cc ii	Red	High
Rr Cc ii	Red	High
rr CC ii	Yellow	High
rr Cc ii	Yellow	High
RR CC Ii	Pink	Intermediate
Rr CC Ii	Pink	Intermediate
RR Cc Ii	Pink	Intermediate
Rr Cc Ii	Pink	Intermediate
rr CC Ii	Cream	Intermediate
rr Cc Ii	Cream	Intermediate
RR CC II	White	None
Rr CC II	White	None
RR Cc II	White	None
Rr Cc II	White	None
rr CC II	White	None
rr Cc II	White	None
RR cc II	White	None
RR cc Ii	White	None
RR cc ii	White	None
Rr cc II	White	None
Rr cc Ii	White	None
Rr cc ii	White	None
rr cc II	White	None
rr cc Ii	White	None
rr cc ii	White	None

Control. The use of colored varieties of onion, where feasible, is the best means of control of smudge as well as of neck rot. Where smudge prevails, colored varieties with few, easily sloughing outer scales should be avoided. When it is desired to grow white varieties, certain practices at harvest tend to reduce the amount of smudge. Protection from rain after harvest and prompt curing are necessary. Since white-onion sets are most seriously damaged by smudge, and since their value is greater than that of large bulbs used for culinary purposes, more elaborate curing procedures are justified. Forced circulation of warm air (up to 48°C.) through the sets stacked in shallow slatted crates, until the outer scales are thoroughly dry and the sets "rattle" on handling, reduces the amount of postharvest development. Storage just above 0°C. is desirable.

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BEAN ANTHRACNOSE

Although the pathogen was actually collected by mycologists as early as 1843, the first description of bean anthracnose, in 1875, was based on specimens originating in Bonn, Germany. It is a major disease of dry and snap beans (*Phaseolus vulgaris* L.) in many parts of the world. Since about 1920 its importance in the United States has declined to a minor role because of the widespread use of anthracnose-free seed. While the disease has been most severe on *P. vulgaris*, it also affects small lima bean (*P. lunatus* L.), large lima bean (*P. limensis* Macf.), scarlet runner bean (*P. multiflorus* Willd.), tepary bean (*P. acutifolius* Gray var. *latifolius* Freeman), mung bean (*P. aureus* Roxb.), cowpea (*Vigna sinensis* Savi.), kudzu bean (*Dolichos biflorus* L.), and broad bean (*Vicia faba* L.) (6, 21).

Symptoms. The disease appears on all above-ground parts of the plant but rarely on the roots (Fig. 77). The lesions are characteristically

dark brown, while in moist weather pinkish spore masses may be present on the surface. Elongate, more or less angular spots appear on the veins on the lower side of the leaf, spreading slightly into surrounding tissue

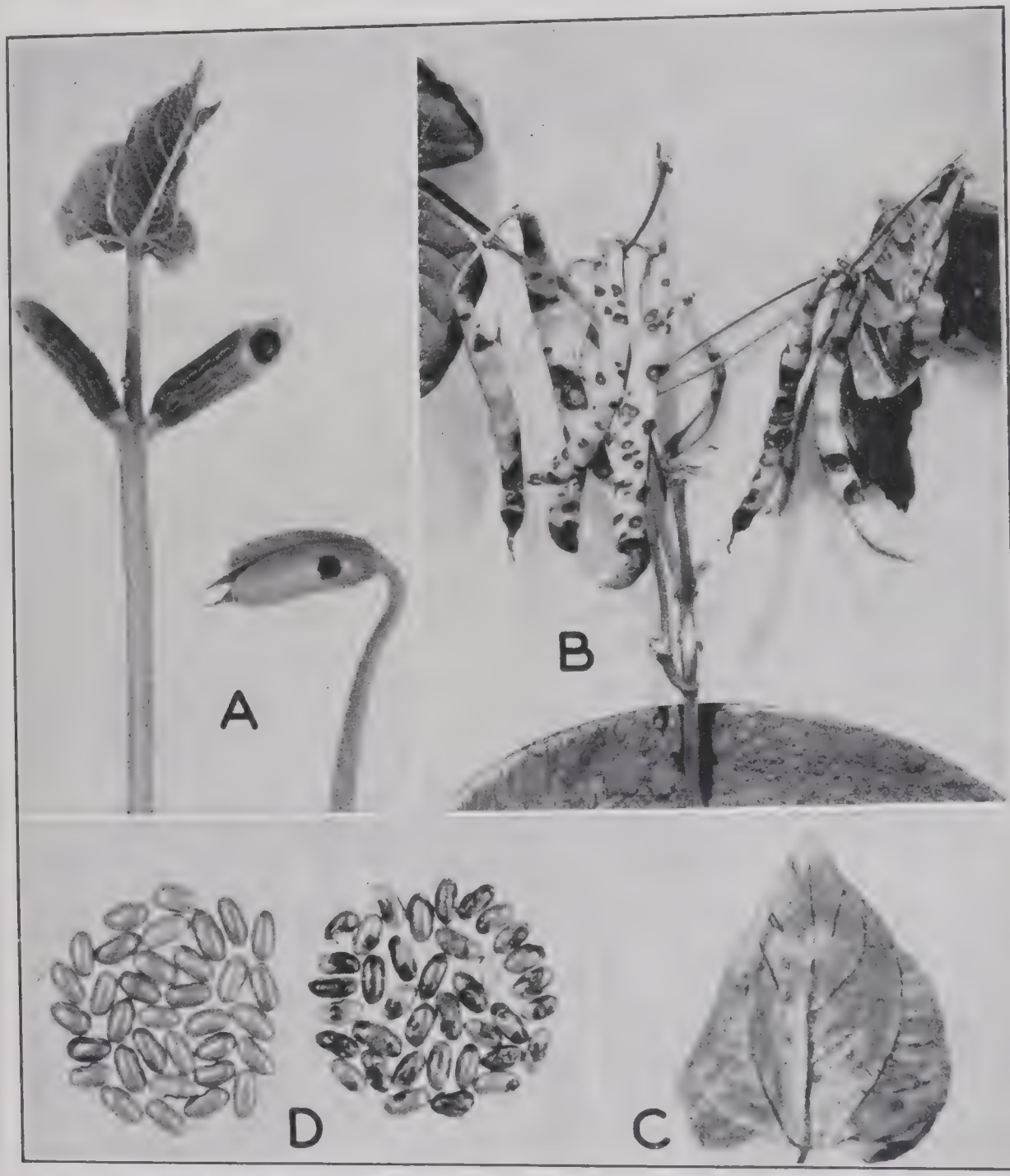

FIG. 77. Bean anthracnose. *A*, lesions on cotyledons growing from infected seeds; *B*, plant with infected pods; *C*, lesions on the leaf characteristically centered on the veins; *D*, uninfected and infected seeds of bean. (*Photographs by R. H. Larson.*)

and eventually appearing on the opposite side. Petioles and stem may also bear lesions, while in the hypocotyl they often extend to deep cankers which result in collapse of the plant. Affected seeds bear lesions colored

in various shades of brown, while in plants which grow from such seeds blackened cankers appear on the cotyledon. The most striking phase of the disease is that on the immature pod. Lesions develop from small brown spots which enlarge quite rapidly up to a diameter of 1 cm. They become dark brown to black in the center and light brown to pinkish at the border. As the pod matures, the colors lighten and the edges of the lesions are raised.

The Causal Organism. *Colletotrichum lindemuthianum* (Sacc. & Magn.) Briosi & Cav., 1889.

Synonymy:

Gloeosporium lindemuthiamun Sacc. & Magn., 1878.
Colletotrichum lindemuthianum (Sacc. & Magn.) Scribner, 1889.
Gloeosporium lindemuthianum Sacc. & Magn. forma *foliicolum* Allesch., 1894.
Colletotrichum lindemuthianum (Sacc. & Magn.) Cav. f. *brachysporum*, 1904.
Glomerella lindemuthianum (Sacc. & Magn.) Shear, 1913.

The mycelium is branched, septate, hyaline at first, becoming dark-colored with age. On culture media, conidia may form singly at the ends of hyphal threads. After constriction of the wall the spore is pushed aside as the hypha elongates and repeats the process. When stromatoid masses form, acervuli are produced upon them, and on the host they form on stromata below the cuticle, rupturing the latter if it is still present on the lesion. Conidia, which are borne acrogenously on short conidiophores, form successively under favorable conditions until pinkish spore masses appear on the surface. Setae occur sparingly. Conidia (4.4 to 5.3 by 13 to 22 μ) are continuous, hyaline, oblong, cylindrical, with ends rounded or somewhat pointed at one end; a clear vacuole-like body often occurs near the center. One to four, usually two, germ tubes arise from a conidium. Appressoria are formed at the tips of germ tubes, especially when they come into contact with the host surface. An ascigerous stage of the fungus in pure culture was described by Shear and Wood (20), but this has never been confirmed and may have been the result of contamination.

Colletotrichum truncatum (Schw.) Andrus & W. D. Moore is a distinct pathogen of bean (1).

Disease Cycle. The incitant lives from one growing season to the next on infected plant debris and on seed. When infected seed germinates, lesions appearing on the cotyledons serve as the source of secondary inoculum. The spores are almost entirely water-borne. Primary leaves and the hypocotyl are foci of secondary infections.

Penetration occurs by means of an infection peg which forms on the side of the appressorium in contact with the host, and it penetrates the cuticle by mechanical pressure. Unlike those of the onion-smudge organism (p. 274) the hyphae do not gelatinize the subepidermal wall but do penetrate the epidermal cell promptly and may enlarge to occupy the entire cell lumen. Leach (12) refers to this stage as "primary mycelium." Hyphae pass through small apertures in the walls, invading several cells during the first 48 hr. after penetration. The surrounding tissue next becomes water-soaked, and "secondary mycelium" consisting of relatively narrow strands transverses cell walls rapidly and with little or no enlargement or bending. The cuticle and epidermis of highly resistant varieties are invaded in a similar manner, but the fungus seldom advances beyond one or two cells, and these die promptly. Parris (15) found that infected leaves assimilated 24 per cent less carbon dioxide than comparable healthy leaves.

Spattering and wind-blown rain are the chief means of local dissemination of the pathogen. Lauritzen (10) found that relative humidity of 92 per cent or higher was necessary for infection, the optimum being close to 100 per cent. No infection occurred above 27°C., and it was prevented or very markedly inhibited at 13°. Abundant infection occurred at 17°. Cultivation of bean fields while rain or dew is present on the foliage provides another important means of dissemination through contact of spore masses with man, animals, and implements. Rainy weather at frequent intervals is essential for build-up of an epidemic.

Anthracnose may develop on snap beans in transit. In such cases infection takes place in the field about the time the beans are picked, and incipient lesions are not visible as they are packed. Lauritzen *et al.* (11) found that symptoms appeared on pods in 5 days at 22°, 25°, and 27°C.; in 7 days at 15.5° and 17.5°; in 9 days at 12°; in 12 days at 10°; in 14 days at 7°.

Beginning about 1920, when production of bean seed began to expand in semiarid irrigated areas of the western United States, it was soon noted that the disease did not develop in that region, owing to lack of sufficient rainfall during the growing season to disseminate conidia. Western-grown seed has continued to be practically free of the anthracnose organism.

Varietal Resistance. Differences of bean varieties in resistance and susceptibility to anthracnose have been noted since as early as 1883. In 1911 Barrus (3) reported that varieties responded differently to different isolates of the fungus. He distinguished two physiological races, which were named *alpha* and *beta*. In 1923 Burkholder (8) distinguished a third strain, *gamma*. In the same year Leach (12) distinguished 8 races, and Schreiber (18, 19) in Germany described 34 races. In each case the races were defined on the basis of host reaction over a selected group of horti-

cultural varieties of bean. Other European investigators have found numerous physiological races, some of which appear to be similar to those found in the United States. Andrus and Wade (2) in 1942 defined a race from North Carolina as *delta*.

McRostie (13, 14) in five intervarietal crosses involving six varieties, found resistance to *alpha* to be controlled by a single dominant gene. When a mixture of *alpha* and *beta* was used as inoculum, dihybrid ratios were secured, indicating that an independent gene for each race was responsible for resistance. Burkholder (8) found, in a cross between two varieties, that resistance to *gamma* was controlled by still a third dominant gene. Schreiber (18, 19), using chiefly two intervarietal crosses involving three parental varieties, found that not less than eight pairs of genes were involved. Andrus and Wade (2) used some 30 intervarietal crosses and six physiological races. In crosses of resistant with susceptible varieties and of resistant with intermediate resistant (tolerant) varieties, resistance was always dominant, and in crosses of susceptible with tolerant, susceptibility was dominant. Thus, the inheritance of resistance to anthracnose involves a large number of genes when a large number of varieties are looked into. Andrus and Wade (2) offered a hypothesis in which, for the parents and hybrids they inoculated with *beta*, *gamma*, and *delta*, 10 genes in three allelic series satisfied the data.

Control. While the complexity of races of the pathogen and of resistance genes in the host makes a breeding program for control through resistance a very involved one, the problem does not offer any very serious difficulty. Such a program has not been followed in the United States because more simple control measures may be applied. Rotation of 2 to 3 years and field sanitation are sufficient to eliminate the fungus on overwintering debris. Procurement of western-grown seed prevents introduction by this avenue. Cultivation when vines are dry is essential if the disease is present. Protective spraying has been suggested, but if the fungus is eliminated by rotation, sanitation, and clean seed, there should be no need to put other measures into practice.

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BLACKLEG OF CRUCIFERS

The blackleg disease of cabbage is of major importance. It may affect cauliflower, broccoli, kohlrabi, kale, Brussels sprouts, turnip, rutabaga, and Chinese cabbage. The fungus was first reported in Germany in 1791 on dead cabbage stems. It was first described on living plants in France in 1849. The phase on rutabaga was first described in Denmark in 1897. The disease on cabbage was reported in the United States in 1910. In the British Isles, Scandinavia and New Zealand, the disease is most important on turnip and rutabaga. In the United States and Australia it is common on cabbage. Rose (12) reported it to be destructive on rutabaga in Minnesota in 1946.

Symptoms. On cabbage and other members of the cabbage tribe, the earliest conspicuous symptoms occur in the seedbed 2 or 3 weeks before transplanting. Spots appear on the leaves as inconspicuous, indefinite pallid areas that gradually become well-defined spots with ashen-gray centers, in which the pycnidia are scattered irregularly (Fig. 78). On stems the spots are more linear and are often surrounded by purplish

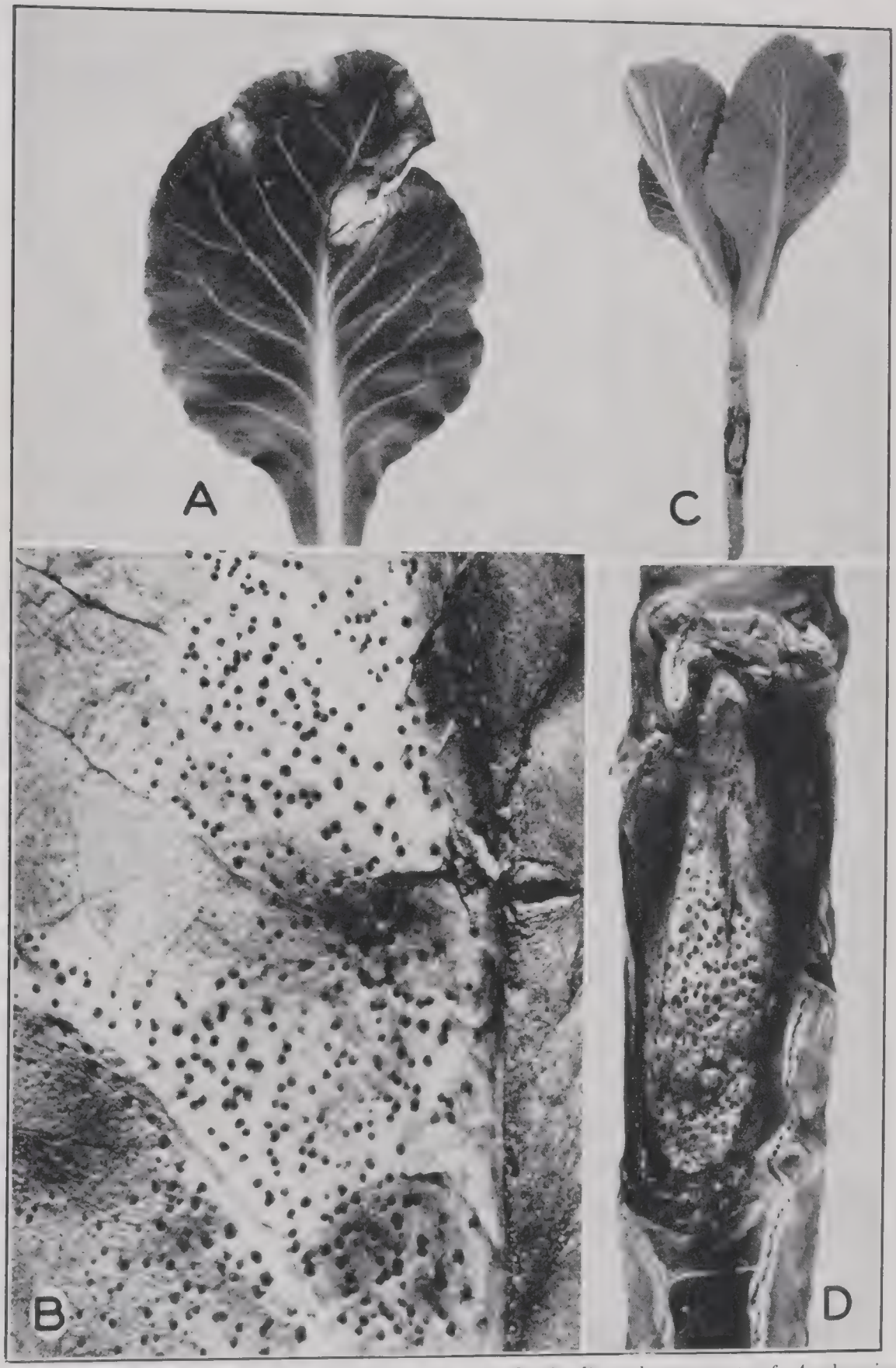

FIG. 78. Blackleg of cabbage. *A*, lesion on leaf; *B*, enlargement of *A* showing pycnidia embedded in the host tissue; *C*, lesion on stem; *D*, enlargement of *C*. (*Photographs from R. H. Larson.*)

borders. Stem lesions at the soil line usually extend to the root system, causing dark cankers in which pycnidia are less common until after the plant is dead. The fibrous root system may be destroyed gradually, although new roots sent out above the lesion may serve to keep the plant alive. Often, badly affected cabbage plants survive until fair-sized heads are formed, when they topple over as the increasing top weight overbalances the gradually decaying root anchorage. Many plants wilt abruptly and die. The wilting leaves tend to remain attached to the stem, instead of dropping by the premature formation of the abscission layers, characteristic of yellows and black rot. As root corrosion progresses, the still-turgid plants often show a reddish color beginning at the margins of the outer leaves. Other types of root injury and phosphorus deficiency may cause similar coloration.

The fungus may invade heads in storage, but it does not cause a serious decay. It affects branches, leaves, and siliques (seed pods) of seed plants, and it invades and causes shriveling of the seeds.

On rutabaga and turnip seedlings, lesions are not unlike those on young cabbage. As the fleshy roots form, they are also affected either before harvest or after they are placed in bins or pits. The infection causes a canker of growing roots and a dry rot in storage. The surface of the decayed tissue may bear pycnidia in abundance.

The Causal Organism. *Phoma lingam* (Fr.) Desm., 1849.

Synonymy:

Sphaeria lingam Tode, 1791.
Phoma lingam (Tode) Desm., 1849.
Phoma siliquastrum Desm., 1849.
Phoma oleraceae Sacc., 1880.
Phoma brassicae Thuem., 1880.
Phoma napobrassicae Rostrup, 1894.
Phoma lingam napobrassicae (Rostr.) Grove, 1935.

Mycelium is septate, branched, hyaline when young, becoming dark-walled with age. Pycnidia vary widely in size and shape between strains and within certain strains. They are generally flask-shaped, usually dark-colored, sometimes with beaked or papillate ostioles. The pycnospores are hyaline, nonseptate, 1 to 2.5 by 3 to 6 μ.

Rostrup (13) considered the form on rutabaga and turnip as distinct and gave the pathogen specific rank (*P. napobrassicae*). Cunningham (5) noted considerable variability in isolations from turnip, rutabaga, and cabbage and concluded that all came within the limits of the original species of Tode. Pound (10) found a form on Chinese cabbage in the Puget Sound area which was distinct in its pathogenic properties but again

within the wide range of variability of forms isolated from cabbage and other crucifers.

The pycnospores are produced in abundance within the pycnidium and on culture medium emerge to form pinkish masses. In nature the spores accumulate within the pycnidium, drying down in a gelatinous matrix. When dew or rain is present, the matrix absorbs water rapidly, and the pycnospores expand in long coils consisting of spores and gelatinous matrix, the latter dissolving in water to release the spores, which are disseminated almost entirely by water (Fig. 79).

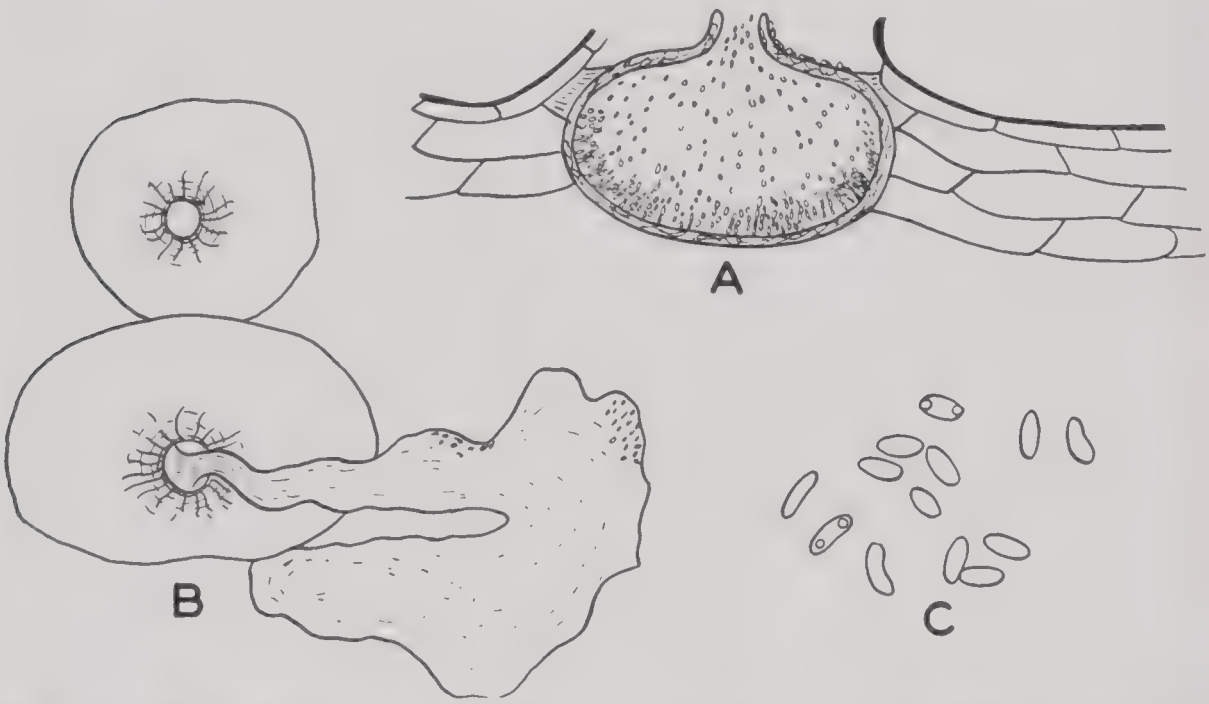

FIG. 79. *Phoma lingam*. *A*, cross section of pycnidium in host tissue; *B*, pycnidia showing gelatinous cirri containing the pycnospores; *C*, pycnospores. (*After Manns.*)

Disease Cycle. The organism lives over for one or more seasons within the seed coat of infected seed or in diseased plant debris. When the seed is infected and still germinable, the seed coat is commonly carried above ground on one of the cotyledons as the plant emerges. Direct infection of this cotyledon follows, and it may be killed as the fungus proceeds to invade the hypocotyl. Such seedlings are usually killed early and are usually unnoticed in the seedbed, although the fungus fruits upon them and provides secondary inoculum. The pycnospores are discharged only in the presence of moisture, and they are disseminated entirely by spattering rain. The development of secondary infection within the seedbed is therefore dependent upon frequent rainy periods, and the epidemiology of the disease is influenced chiefly by rainfall. If dissemination occurs in the seedbed, plants may become generally infected before transplanting.

Surface drainage water may carry spores to the seedbed from infected debris in fields of the previous season. Much of the development on transplanted plants is due to incipient infection which occurred in the seedbed.

Very mildly affected heads saved for seed production carry the fungus over winter and provide inoculum which infects the seedstalks.

In the Pacific coast regions the lack of rainfall during the seedbed season is effective in preventing development of blackleg. This was shown in a series of studies by Walker (14, 16), and Pacific coast–produced crucifer seeds have been found to be generally free from *Phoma lingam* as well as from the black-rot organism. Pound (10) found the Chinese cabbage strain common in the Puget Sound area, although this is very mildly pathogenic on cabbage. Pound and Calvert (11) reported isolation of the blackleg strain, severely pathogenic on cabbage, in lots of cabbage seed grown in the Puget Sound area in 1946.

Variability of *P. lingam* was studied extensively by Pound (10). Calvert *et al.* (3) induced mutations of various isolates by chemical and physical means. They secured many mutants but did not secure any from the Chinese cabbage strain which resembled the severe blackleg strain in pathogenicity. Calvert and Pound (2) found two distinct strains which, without anastomosis of mycelia, participated in formation of pycnidia, each thallus abstricting its own pycnospores within a single fruiting body. Such pycnidia are known as **symphogenous.**

Control. It is essential to have a rotation of at least 3 years between susceptible cruciferous crops. This is particularly important in respect to the seedbed of transplanted crops. Rotation is of no avail if debris from nearby fields is allowed to become a source of inoculum transmitted by means of surface drainage water, cultivating equipment, or roving animals. Noninfected seed is imperative. Pacific coast seed has been generally regarded as *Phoma*-free but findings of Pound and Calvert (11) show this is not always so. Chemical dust and liquid treatments are not effective in ridding seed of *Phoma*. The only reliable treatment is with hot water at 50°C. for 30 min. Many lots of seed are markedly reduced in germinative capacity by this treatment, particularly 2- or 3-year-old lots or those which are weak for one or another reason. It is advisable, therefore, to determine in advance whether the seed lot in question will stand treatment by making a preliminary run on a few hundred seeds. If the seed will not stand treatment, it is advisable to secure seed that will.

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DIPLODIA DISEASE OF CORN

The *Diplodia* disease of corn was described as an ear rot in the United States in 1909 by Heald *et al.* (4) in Nebraska and by Burrill and Barrett (1) in Illinois. It has since become known as a seedling blight, root rot, stalk rot, and ear rot. It is most serious in the southern half of the corn belt, especially in Ohio, Indiana, Illinios, and Iowa. It is usually least serious in the drier areas of the belt to the west and in the cooler areas to the north. The disease is often part of a complex in which other pathogens are involved, particularly *Gibberella* spp. and *Nigrospora* spp. The fungus has a world-wide distribution on corn.

Symptoms. Stalks are rarely infected by *Diplodia* or other stalk-rot fungi before the ears have reached the dough stage. Root infections, which spread into the crown and lower stalk tissues, comprise the most serious aspect of the disease since they result generally in chaffy ears,

broken stalks, and premature death (Fig. 80). The development of the disease following root infection is rapid. The green color of the stalks fades quickly, the leaves suddenly develop an appearance like that caused by early frosts, and the plants soon die. In addition to a shredded condition of the pith tissues in plants which die prematurely with stalk rot, a drooping of the tassel is characteristic, apparently an effect of acute wilting which precedes death of the plants. Infection at lower internodes from air-borne spores which have lodged inside the leaf sheath usually results in a softening and a brown discoloration at the node which spread into adjoining tissue. The black pycnidia of the pathogen may appear on all above-ground lesions. They develop most abundantly late in the fall and on old stalks the following spring. Ears are attacked usually through the butt from infections which occur on the ear shank. The white mycelium appears between the kernels and grows into the husk, cementing it to the kernels. When ears become completely rotted from early infections, the leaves of the entire plant turn purplish red and die prematurely. Many ears are not visibly infected at harvest, but progress of the disease in storage may lead to all degrees of kernel infection. Mildly infected seeds may show no signs of disease, but upon germination the mycelium of the pathogen develops rapidly. When planted in cold soil, seeds decay and preemergence death of seedlings commonly results. In warm soils better stands result, but many of the seedlings are stunted. These usually have brown cortical lesions on the internode between the scutellum and the coleoptile (mesocotyl), and seminal roots are frequently destroyed.

The Causal Organisms.

1. *Diplodia zeae* (Schw.) Lév., 1848.

Synonymy:

Sphaeria striaeformis var. γ Schw., 1822.
Sphaeria zeae Schw., 1832.
Sphaeria maydis Berk. 1847.
Sphaeria (*Hendersonia*) *zeae* (Schw.) Curr., 1859.
Hendersonia zeae (Curr.) Hazsl., 1873.
Diplodia maydis (Berk.) Sacc., 1884.
Dothiora zeae (Schw.) Bennett, 1888.
Macrodiplodia zeae (Schw.) Petr. & Syd., 1923.
Phaeostagnosporopsis zeae (Schw.) Woron., 1925.

2. *Diplodia macrospora* Earle, 1897.

The pycnidia are globose, flask-shaped, or irregular in shape. Conidia are ovate, olivaceous, two-celled, straight to slightly curved with rounded to bluntly tapered ends. The conidia of *D. zeae* measure 6 by 25 to 30

μ; those of *D. macrospora*, 6 to 8 by 70 to 80 μ. Slender, thread-like, hyaline scolecospores are formed. *D. zeae* is the most common; *D. macrospora* is found chiefly in the southern states. Two other species (*Physalospora zeicola* Ell. & Ev. and *P. zeae* Stout), which differ somewhat in the imperfect stage from *D. zeae* and *D. macrospora* and which have ascigerous stages, occur in southeastern and central United States (23).

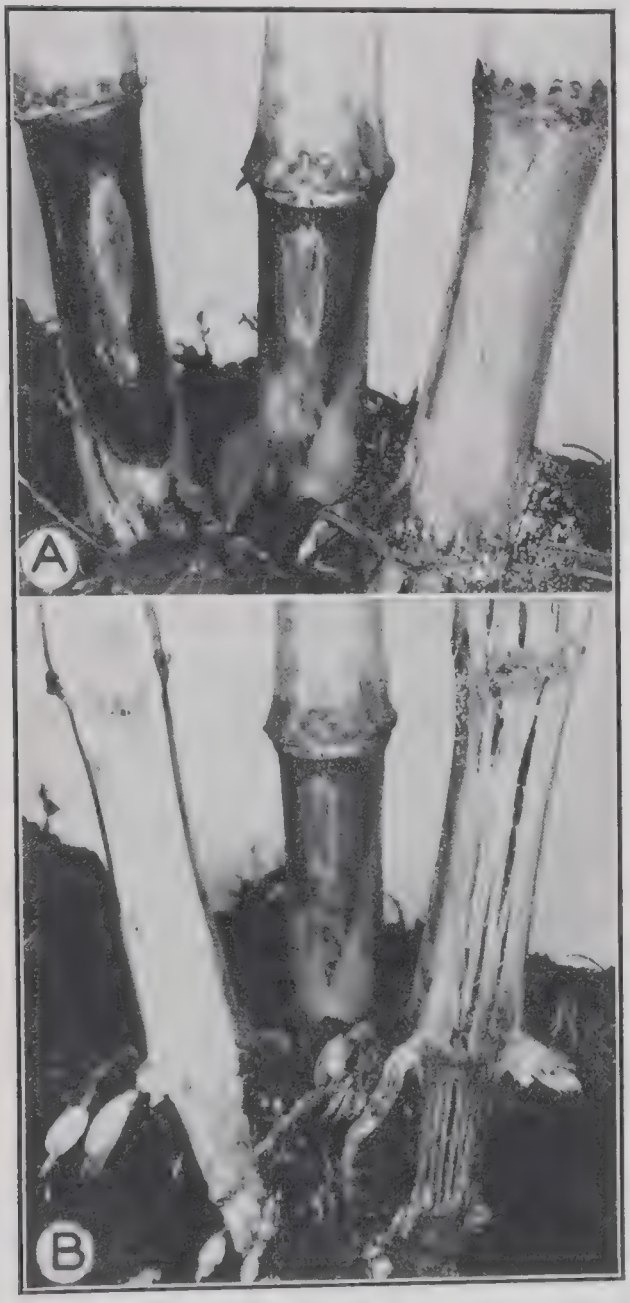

FIG. 80. *Diplodia* stalk rot of corn. *A*, lesions at bases of stalks, one showing pycnidia; *B*, cross sections of healthy and diseased stalks. (*Photographs from P. E. Hoppe.*)

The physiology of the *D. zeae* and *D. macrospora* has received considerable study. Wilson (25) showed that *D. zeae* grows well on synthetic media composed of highly purified materials, while *D. macrospora* does not. The filtrate from cultures of *D. zeae*, however, contains the growth-promoting substance needed for growth by *D. macrospora*. The characteristics of the substance suggest that it is biotin. It occurs in green cornstalks and as an impurity with certain carbohydrates. This phenomenon may account for the slower growth of *D. macrospora* on some media reported by Kinsel (14) and Hoppe (7). Hoppe (7) found that when colonies of certain isolates of *D. zeae* were grown on an agar plate, their hyphae intermingled freely as the colonies merged. Between other isolates a pronounced aversion occurred as the colonies approached one another. Growth of the hyphal tips ceased, and there was an apparent dying back of the mycelium. This was not characteristic of the reaction of a staling product commonly observed among fungi. When two averting strains were inoculated into a corn ear, one took precedence, and after the ear had decayed, only one

strain was recovered. A similar aversion was found between *D. zeae* and *D. macrospora*. What appears to be a typical staling product was observed by McNew (17) and was studied further by Kent (13). It is a nonvolatile, thermolabile substance liberated by the mycelium after 5 to 11 weeks in culture, and it was also found in infected parts of the host plant.

Disease Cycle. The fungi overwinter in seed, diseased stalks, and infested soil. The cardinal temperatures for *D. zeae* on culture media are minimum, 10 to 15°C.; optimum, 28 to 30°; maximum, 35 to 40°. In general, more seedlings are killed at low than at high soil temperatures. This is due primarily to the fact that corn seedlings grow most rapidly in warm soil, and the effect of the pathogen is greatest when the seedlings are growing slowly. Pycnospores are air-borne. Infection may occur through the silk, at the larger nodes between leaf sheath and stalk, and in the shank. Invasion of the ear is by way of the shank. Seedlings are infected primarily by the inoculum introduced in the seed. Root and crown infections may result from inoculum in seedlings which have recovered, but they occur primarily as a result of later invasion by the fungus in the soil. The stalk tissue increases in susceptibility to the pathogen after pollination. Johann and Dickson (12) found an ether-soluble substance in cornstalks which delayed growth of the pathogen. This growth-retarding substance declined in amount after pollination and in correlation with increase in susceptibility. Differences in rate of decline were found between lines of corn showing different degrees of susceptibility. The authors suggested that differences in resistance might be due to differences in development of the host with relation to the inhibitive substance.

Control. Rotation and sanitation are advisable precautionary methods but they cannot be relied upon for effective control when seed is infected. Seed treatment is essential to control this and other seed-borne pathogens and to protect the seed from soil-borne organisms. Semesan Jr. and Arasan (see Chap. 16) have been used widely for corn-seed treatment. They are superior to Spergon, since they are more effective at low temperatures than the latter and act as disinfectants as well as protectants. Semesan Jr., while it is superior to Arasan as a disinfectant, has the disadvantage of being more phytotoxic when seeds have breaks in the pericarp. Arasan is more widely used because it is a good protectant, is nearly as good a disinfectant as Semesan Jr., and is less phytotoxic than the latter (8, 9).

It has been shown that inbred as well as hybrid lines of corn differ markedly in resistance to the crown-rot and stalk-rot phases of the disease. *Diplodia* resistance is, therefore, one of the objectives in breeding programs in the corn belt. It is possible to avoid hybrids which are particularly

susceptible to stalk rot, and the use of the proved resistant ones is important in areas where the *Diplodia* disease is likely to be destructive (5, 21, 22).

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LATE BLIGHT OF CELERY

Late blight is one of three destructive foliage blights of celery. The other two are early blight (*Cercospora apii* Fres.) and bacterial blight (*Pseudomonas apii* Jagger). All three may occur simultaneously. Late blight was first recorded in Italy in 1890. It was reported the next year in Delaware, in Denmark in 1893, in France in 1894, in Germany in 1896, and in the British Isles in 1910. Since the pathogens are seed-borne, the disease has become world-wide in occurrence. It reduces yield and blemishes the edible petioles. When control measures are not applied, losses may become very heavy. The late-blight pathogens are restricted to celery (*Apium graveolens* L.) and celeriac (*A. graveolens* var. *rapaceum* DC.).

Symptoms. There are two species of *Septoria* which incite late blight. The symptoms differ chiefly in the size of the leaf spot produced, and the two forms are referred to as the small-spot form and the large-spot form. In the small-spot form small chlorotic spots appear, which become necrotic, beginning at the center (Fig. 81). The necrotic spots remain small, seldom exceeding 3 mm. in diameter unless they coalesce on the fleshy petiole. While the necrotic areas have a definite margin, they are often surrounded by a chlorotic zone which merges gradually into normal green tissue. The chlorotic areas commonly coalesce to affect the entire leaf. Fruiting bodies may appear even before the first sign of chlorosis, and they may continue to appear outside of the necrotic area. As the tissues in the latter die, the pycnidia become very conspicuous as small black bodies which are numerous, closely crowded, and visible to the naked eye. The small-spot form is by far the most prevalent and important.

The large-spot form first appears as a small chlorotic fleck. The tissue collapses in all directions until a spot 3 to 10 mm. in diameter with a definite border is formed. Pycnidia appear only near the center of the spot and are scattered and sparse. The necrotic tissue is brown to reddish brown toward the center and darker reddish brown at the border.

Foster and Weber (3) reported that the two species produced identical lesions on petioles. Cochran (1), however, failed to produce lesions on the petiole with the large-spot form.

The Causal Organisms.

Small-spot form: *Septoria apii-graveolentis* Dorogen, 1915.

Synonymy: *Septoria apii* var. *punctiformis* Laibach, 1921.

Large-spot form: *Septoria apii* (Briosi & Cav.) Chester, 1891.

SYNONYMY:

Septoria petroselini Desm. var. *apii* Briosi & Cav., 1890.
Phlyctaena magnusiana (Allesh.) Bresadola, 1892.
Septoria apii var. *maculiformis* Laibach, 1921.

The pycnidia of *S. apii-graveolentis* are 73 to 147 μ in diameter, with ostioles one-third to one-half the diameter of the pycnidium. Those of

A B

FIG. 81. Late blight of celery. The small-spot form on leaf (*A*) and petiole (*B*). (*B, courtesy of U.S. Department of Agriculture.*)

S. apii are 65 to 95 μ in diameter with ostioles less than one-fourth the diameter of the pycnidium. The pycnidia of *S. apii-graveolentis* are usually crowded and sometimes fused; those of *S. apii* are nearly spherical and are always borne singly. Conidia of both species are long, slender, filiform, hyaline, and faintly septate. MacMillan and Plunkett (7) state that no true septa are present and that what appear to be walls are lines between abutting plasma membranes of contiguous "cells" which give, through differential light diffraction, the appearance of septa when viewed

under the microscope. Conidia of *S. apii-graveolentis* are flexuous, blunt at the ends, granulose, several-septate, 22.5 to 58.5 by 1.5 to 3.0 μ. Those of *S. apii* are straight to slightly curved, minutely granular, several-septate, 13.5 to 34.2 by 1 to 2.5 μ (Fig. 82).

Since the differentiation of the two species was not made until the publication by Dorogen in 1915 and since the work was not brought to general

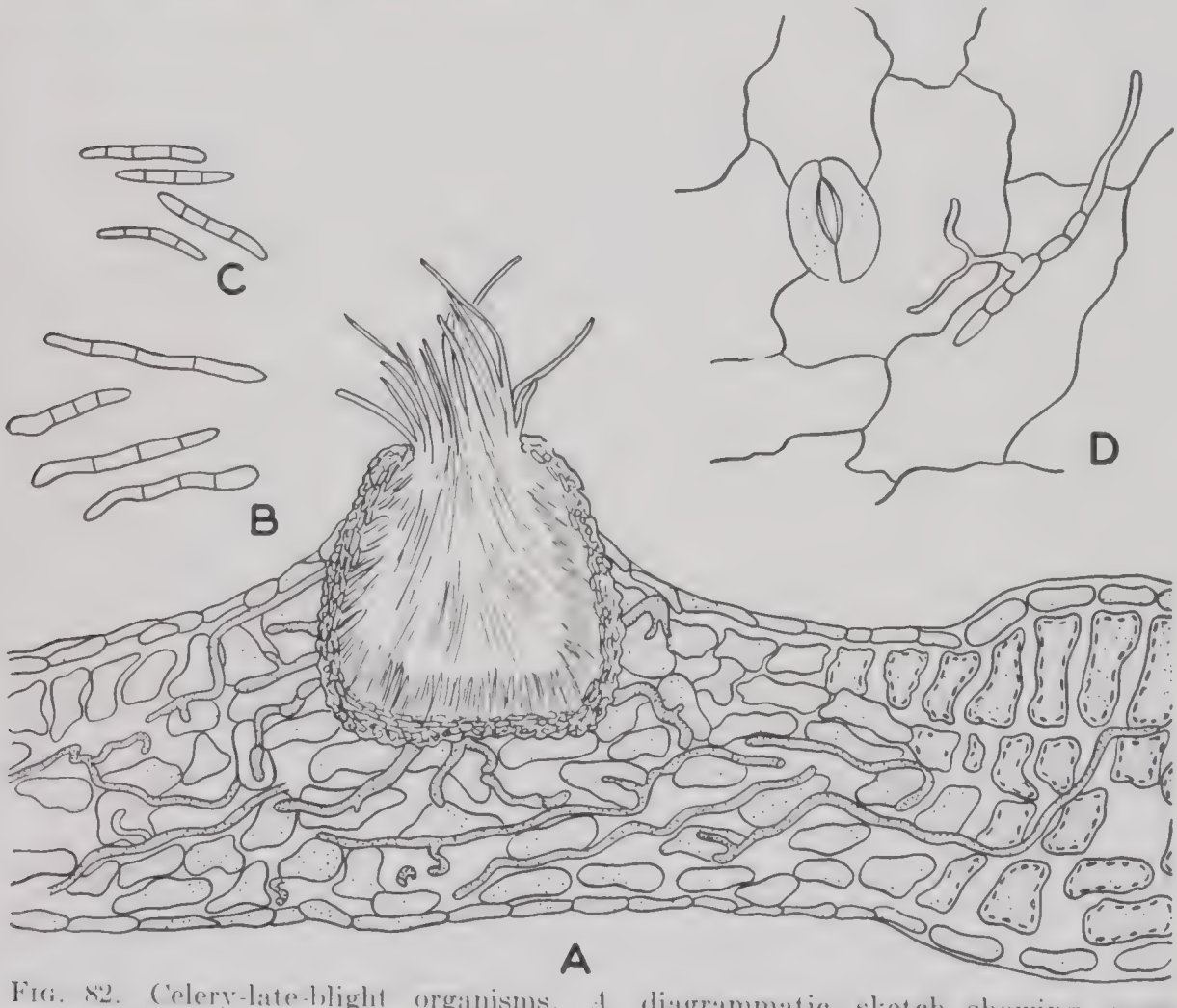

FIG. 82. Celery-late-blight organisms. *A*, diagrammatic sketch showing cross section of pycnidium and mycelium in celery leaf; *B*, pycnospores of *Septoria apii-graveolentis*; *C*, pycnospores of *S. apii*; *D*, direct penetration of celery leaf tissue. (*A after Coons and Levin; B–D after Cochran.*)

attention until the confirmatory investigation of Cochran (1) in 1932, the causal organism was referred to generally until the latter date as *S. apii*. Since the most common species is *S. apii-graveolentis*, much of the literature concerns it, and before 1932 it was usually erroneously referred to as *S. apii*.

Disease Cycle. The information acquired upon the life history of the organisms is concerned almost entirely with *S. apii-graveolentis*. The fungus affects the seeds, causing pycnidial formation in the seed coat and being widely distributed by this means. Survival between crop seasons

is in seed and in crop refuse in both the northern and southern United States. The first appearance of the disease is usually in the seedbed. Dissemination is chiefly by spattering and wind-blown rain. It may be brought about by contact with clothes of workmen, animals, and implements (6). The fungus penetrates the leaf tissue directly.

Conidia of both species germinate over a range of 9 to 28°C., the optimum for *S. apii-graveolentis* being 20 to 25° and that for *S. apii* slightly higher. Humid weather favors dissemination and epidemic development. Late blight develops more rapidly in cool than in warm weather. In the northern states it is more destructive in the latter part of the growing season, as contrasted with early blight, which is favored by warm weather. For this reason the two diseases have acquired their respective common names. In winter-grown celery in Florida the succession is usually in reverse order, late blight appearing first in cool early spring months and early blight developing as the average temperature rises.

Control. Seed treatment with hot water (48 to 49°C., 30 min.) (4) is the only measure which eradicates the fungus from infected seeds. While this practice is not generally used in the United States, largely because inoculum arising from overwintering refuse offsets the benefit derived, there are cases reported which show that seed treatment has a delaying effect on epidemic development (2). Three-year-old seed is recommended because as a rule the fungus is no longer viable after this period.

Sanitary measures designed to reduce the amount of carry-over inoculum are important, but in practice this procedure combined with seed treatment does not assure freedom from late blight.

Control through the application of fungicides is, therefore, an essential and satisfactory control, which applies equally well to early blight and bacterial blight. There has been much experimentation in the evaluation of dusting and spraying methods and of the relative effectiveness of various fungicides. Copper-lime dust is about as effective as Bordeaux mixture spray, but it is more difficult to apply the former with maximum efficiency. When compared with insoluble copper compounds and organic compounds, Bordeaux mixture has in most cases proved to be equal or superior. The standard procedure is, therefore, to use 8-8-100 Bordeaux mixture at intervals of approximately 6 days (8).

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WHITE ROT OF SHALLOT, ONION, AND GARLIC

In 1841, Berkeley described in England a disease of onion which is now referred to as white rot. It seems not to have attracted much attention until early in the present century. Voglino recorded it as a serious disease of garlic in northern Italy in 1902. It has since been regarded as a common and sometimes destructive disease of onion, garlic, shallot, and leek throughout western Europe as well as in Cyprus, Egypt, India, Ceylon, Australia, South Africa, the Canary Islands, Argentina, and Brazil. The first authentic record of white rot in the United States was on onion and garlic collected in northeastern Oregon in 1918. It has since been reported in Virginia (1923), Kentucky (1925), New Jersey (1936), California (1938), and Louisiana (1942). The known host range is onion (*Allium cepa* L.), garlic (*A. sativum* L.), shallot (*A. ascalonicum* L.), leek (*A. porrum* L.), Welsh onion (*A. fistulosum* L.), and wild garlic (*A. canadense* L.).

Symptoms. The disease may affect the plant at any time during the growing period provided that the environment is favorable. The first sign is yellowing and dying back of the leaves, beginning at the tips and progressing downward. The rate of the advance varies with the rapidity of fungus attack, and this in turn depends upon the environment. Young plants may wilt and collapse rapidly. A gradual decline of the plant may continue for some days or weeks. Coincident with early symptoms above ground, evidence of disease appears on the subterranean portions of the host plant. Roots and bases of scales are attacked by the fungus, which becomes conspicuous by an abundance of superficial, white, fluffy mycelium. A semiwatery decay destroys roots and scales. Early in disease development black spherical sclerotia about the size of the head of a pin appear on the surface of, or are embedded within, the decayed tissue. If bulbs or corms are infected late in their development, only incipient decay may have occurred at harvest and the disease continues as a storage rot (Fig. 83).

The Causal Organism. *Sclerotium cepivorum*, Berk., 1841. White, fluffy, branched mycelium is produced in abundance on host tissue and on agar media. Voglino described sporodochia of hyaline conidiophores bearing spherical, hyaline, catenulate conidia (3 to 4.5 μ), and on the basis

of these structures he renamed the fungus *Sphaeelia allii*. Others have found the so-called conidia incapable of germination. They resemble the microconidia characteristic of some species of *Sclerotinia*, in which they sometimes serve as male gametes. Sclerotia are formed abundantly as a rule, but aberrant strains which are largely mycelial have been reported.

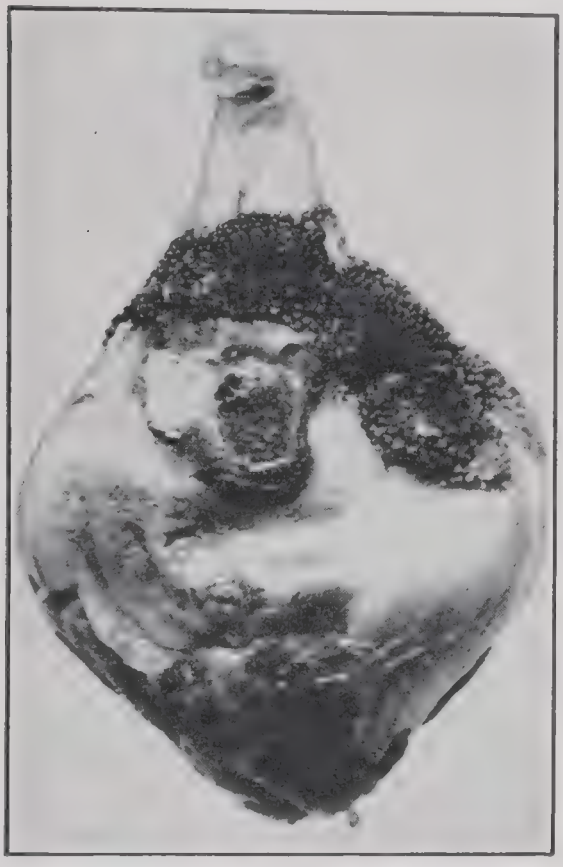

FIG. 83. White rot of onion (compare with neck rot in Fig. 62).

Disease Cycle. It is interesting to note that the pathogen persists for long periods without any known functional sporulating stage. It is distributed over long distances on bulbs, and when once established in favorable soil, it remains viable for several years in the absence of a host. Walker (4) studied the relation of soil temperature and soil moisture to the disease. The temperature range for growth on agar is from about 4 to 30°C., with an optimum at 20 to 24°. In constant-soil-temperature experiments it was found that the disease developed most rapidly between 10 and 20°. At 22° the percentage of infected plants was low, and the rate of disease development was markedly reduced. At 24° and above, the plants remained healthy in heavily infested soil. At relatively constant soil moisture the disease developed much more rapidly in fairly dry soil (40 per cent of the water-holding capacity) as compared with moist soil (60 to 80 per cent) (Fig. 175). It is of particular interest that the disease is practically prevented at the upper end of the optimum temperature range for growth of the fungus in pure culture.

While white rot has become quite common in Europe, it was found by Walker (3) to be rare or absent in the warm onion-growing regions of Spain but severe in the cooler mountainous garlic-producing areas of that country. Being a cool-soil disease, it is of interest that the fungus has become established in the United States only in the southern states and where winter-grown crops are concerned. It is of greatest economic significance on midwinter shallots in Louisiana. In that region Tims (2) has found that early fall plantings escape the disease successfully as compared with midwinter plantings. He also found the disease most severe

in the higher, better drained portions of the field than in lower wetter areas.

Control. In the shallot-growing area of Louisiana the only remedial measure of consequence devised so far is to grow only a fall crop on badly infested areas. While a mercuric chloride drench around plants reduced the disease damage somewhat, it is hardly practicable. Liming of soil to bring the pH up to about 7.0 reduced the disease but did not afford complete control (2). In England applying mercurous chloride to the seed drill affords control (1).

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CHAPTER 8

DISEASES INCITED BY ASCOMYCETES

The Ascomycetes, or sac fungi, are a group containing many thousands of species, which range from pure saprophytes to those which are obligate parasites upon higher plants. The first three orders of the Fungi Imperfecti (p. 244) contain forms which resemble the various imperfect stages of Ascomycetes. In fact, in many cases, species of the latter class have been described originally on the basis of the imperfect form in one or another genus of the Imperfecti and later transferred to the Ascomycetes when the perfect stage was discovered and described at a later date.

As in the Fungi Imperfecti, the mycelium is septate, uninucleate or multinucleate, commonly forming chlamydospores, stromata, and sclerotia. Conidia are usually produced but not in all species. In the sexual stage the distinctive feature is the **ascus,** a sac-like body, in which **ascospores,** typically eight in number, are formed. The asci may arise singly without a distinct fruiting body (Hemiascomycetes) or within one or another type of fruiting body (Euascomycetes). The three major types of fruiting bodies (**ascocarps**) are as follows: **cleistothecium,** a roughly spherical body with no natural opening for the discharge of ascospores; **perithecium,** a globular to flask-shaped fructification body which has an apical pore (**ostiole**) through which ascospores are released; **apothecium,** usually a disk-like or cup-shaped body whose inner (upper) surface consists of a hymenium bearing the asci in a palisade layer, from which the spores are discharged directly into the atmosphere. Cleistothecia and apothecia are ordinarily borne in an exposed position on the thallus. The same is frequently true of perithecia, but in a considerable number of species the latter are embedded in a compact and often massive aggregation of hyphae known as a **stroma.**

While the development of the **perfect (ascocarpic) stage** has been studied in detail in only a relatively few members of the Euascomycetes, it appears that a sexual process is commonly involved in the initiation or early development of the fruiting body. The female organ is a specialized cell or a specialized hypha termed an **ascogonium.** This develops at the end of a branch arising from the ordinary vegetative mycelium or differentiates within a compacted mass of hyphae comprising an ascocarp initial. The ascogonium receives one or more nuclei from a male organ (**antheridium,** or **spermatium**), often by way of a **trichogyne,** a receptive extension of the female organ proper. From the ascogonium arise the **ascogenous hyphae,** which ultimately give rise to the spore sacs. If the ascogonium was initially exposed, a mass of sterile hyphae grow up

from below it about the time of fertilization and form the envelope or supporting structure of the fructification.

Some Ascomycetes are **homothallic,** in which case the fusing sexual structures are borne on the same mycelium; others are **heterothallic,** the cooperation of two compatible thalli being essential for fertilization to be accomplished. In the latter situation the ascogonium receives a nucleus or nuclei of complementary reaction from antheridia, or spermatia, produced on a different thallus. Various substitute sexual processes are encountered among the Ascomycetes. One or both sex organs may be missing and heterothallism still operate through substitution mechanisms. In the ascogonium the female and male nuclei mingle and commonly pass into the ascogenous hyphae in pairs, there to multiply by a type of conjugate division. The tips of the ascogenous hyphae recurve, forming what are termed **crosiers,** and the penultimate cell of each enlarges to become an ascus. Two nuclei are regularly present in the penultimate cell and hence in the young ascus; in heterothallic species these nuclei are known to be of different reaction. As the young ascus enlarges, the nuclei fuse to form a 2n primary ascus nucleus, and there follow three nuclear divisions, one of which is reductional, to give eight *n* nuclei. Around each of the eight nuclei an ascospore is formed by delimitation of the cytoplasm through formation of cell walls. This is usually a process known as **free-cell formation,** in that it involves the appearance of walls within the cytoplasm of the ascus to cut off the spores, leaving the remainder of the cytoplasm outside of the spores but within the ascus. Spores are ejected from the ascus in various ways and give rise to mycelium typical of the species. In many of the Euascomycetes the asci are closely intermingled with numerous sterile hair-like hyphae known as **paraphyses.** These commonly are taller than the asci and in apothecia undoubtedly serve to protect the otherwise exposed spore sacs. At the time of spore discharge the ascus usually elongates, its apex thus being brought above the level of the tips of the paraphyses.

While the pathogenesis of Ascomycetes has much in common with that of the Fungi Imperfecti, there is the addition of the ascospore stage, which, it will be seen, may make the disease cycle more complex. In some cases only ascospores serve as the inoculum; in many both ascospores and conidia serve in this capacity, but their dispersion may be brought about in quite different ways. The ascus stage in some species is an intimate part of the parasitic phase of the pathogen; in other cases it occurs only as a part of a saprophytic stage of the organism.

The subdivisions of the class, including the orders and families from which the pathogens of the diseases treated are drawn, are characterized briefly below:

Hemiascomycetes. Asci borne singly or clustered on the mycelium; no specialized fruiting body present.

Exoascales or Taphrinales. Mycelium poorly developed; budding common; all parasitic on seed plants and ferns.

Taphrinaceae.

Taphrina deformans, (Berk.) Tul., incitant of peach leaf curl.

Euascomycetes. Asci borne in ascocarps.

Plectomycetes. Ascocarps closed (cleistothecia), opening only by weathering; asci globose to broadly clavate.

Erysiphales, or Perisporiales. Parasitic on green plants.

Erysiphaceae, powdery mildews.

Erysiphe graminis DC., incitant of powdery mildew of cereals.

Pyrenomycetes. Ascocarps opening by a small pore or slit; asci clavate to cylindric.

Sphaeriales. Perithecium carbonaceous; wall differentiated from the stroma if present.

Ceratostomataceae. Perithecium free from the substrate, dark-colored, and with long ostiolar beak.

Ceratostomella ulmi Buismann, incitant of Dutch elm disease.

Mycosphaerellaceae. Perithecia embedded in substrate, papillate.

Mycosphaerella pinodes (Berk. & Blox.) Stone, incitant of *Mycosphaerella* blight of pea.

Venturia inaequalis (Cke.) Wint., incitant of apple scab.

Gnomoniaceae. Perithecia embedded in substrate, beaked.

Glomerella cingulata (Stone.) Spauld. & Schrenk, incitant of bitter rot of apple.

Diaporthaceae. Perithecia immersed in pulvinate stromata composed of both fungus and host elements.

Endothia parasitica (Murr.) P. J. Anderson & H. W. Anderson, incitant of chestnut blight.

Hypocreales. Perithecia and stroma bright colored—red, yellow, purple, etc.; wall of perithecium well-differentiated from stroma.

Gibberella zeae (Schw.) Petch, incitant of scab and seedling blight of cereals.

Claviceps purpurea (Fr.) Tul., incitant of ergot of cereals and grasses.

Discomycetes. Ascocarps open, commonly cup- or disk-shaped (apothecia).

Helotiales. Asci inoperculate, *i.e.*, not opening with a lid or transverse slit.

Helotiaceae. Apothecia fleshy, leathery or corneous and usually superficial.

Sclerotinia fructicola (Wint.) Rehm, incitant of brown rot of stone fruits.

Sclerotinia sclerotiorum (Lib.) DBy., incitant of drop or watery soft rot of vegetables.

PEACH LEAF CURL

This disease has been recorded in Europe and in the United States since early in the nineteenth century. It is distributed throughout the world wherever peach is grown. Leaf curl is of greatest economic importance

in the United States in the Great Lakes region and in the valleys west of the Cascades and Sierra Nevada ranges in Washington, Oregon, and central and northern California. Peach is the chief economic host. It also occurs on nectarine and peach almond. Other species related closely to the pathogen occur on some other stone fruits.

Losses at one time were very heavy in the United States, but this disease is one which is rather easily controlled when proper protective measures are taken.

FIG. 84. Peach leaf curl. (*Photograph from A. J. Mix.*)

Symptoms. The disease first appears, although relatively inconspicuously, in the early spring as the leaves begin to unfold. The blade becomes thickened and puckered along the midrib, and curling begins. As the disease progresses, the leaf tissue becomes yellowish and shows some reddish color. It eventually takes on a silvery appearance on the upper surface. Leaf and petiole may both be involved in the curling. The leaf becomes thicker than normal in the process, and the tissues become noticeably brittle (Fig. 84). Affected leaves die and drop prematurely while otherwise dormant buds become active to produce new leaves. Twigs may also be affected. They become pale green to yellow, swollen, stunted and sometimes exude gummy material. Infected twigs usually die. Flowers and fruits may be infected, but they drop promptly, and affected fruits are seldom noticed.

The Causal Organism. *Taphrina deformans* (Berk.) Tul., 1866.

SYNONYMY:

Ascomyces deformans Berk., 1857.
Ascosporium deformans Berk., 1860.
Exoascus deformans (Berk.) Fkl., 1869.

The mycelium occurs intercellularly in the host tissue. It is branched, and each cell contains one or more pairs of nuclei which divide conjugately (Fig. 85). There is no ascocarp. Ovoid, pyriform, or dome-shaped ascogenous cells appear beneath the cuticle of the leaf, most often on the upper surface, each with dense cytoplasm and two nuclei. Fusion takes

place in the ascogenous cell to form the diploid nucleus, which contains eight chromosomes. The cell then elongates, and its wall becomes thinner, while the nucleus goes through one vegetative division. One nucleus moves to the distal end of the cell while the second nucleus is contained in a basal cell cut off by a septum. The distal cell becomes the ascus. Three successive divisions of the nucleus occur; during this, reduction takes place to the haploid stage with four chromosomes. Ascospores are formed by the union of vacuolar membranes rather than by the free-cell formation described for some other Ascomycetes. Usually eight unicellular ascospores are formed, but occasionally there may be as few as two. In any case each spore is uninucleate (Fig. 86).

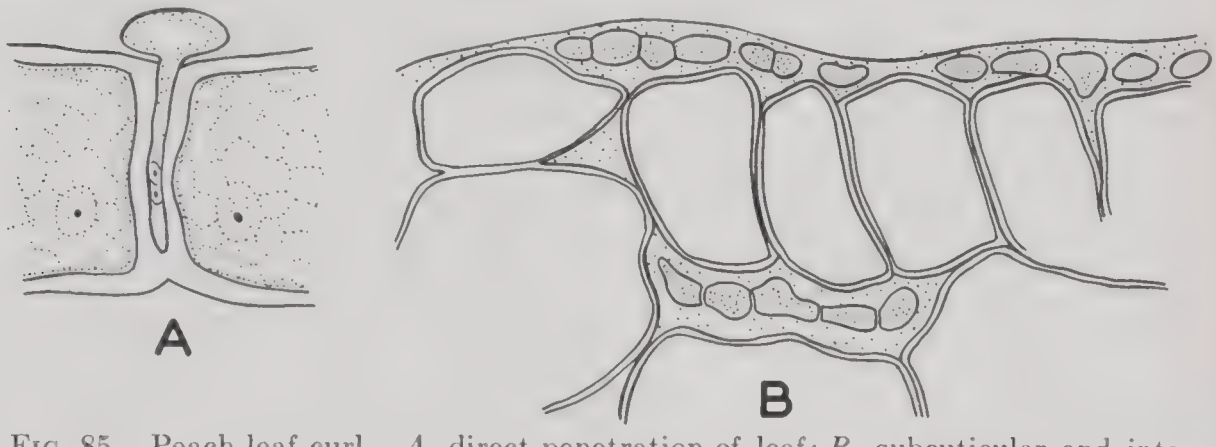

FIG. 85. Peach leaf curl. *A*, direct penetration of leaf; *B*, subcuticular and intercellular mycelium. (*A after Mix; B after Pierce.*)

Ascospores commonly germinate by budding to form a series of uninucleate thin-walled secondary spores which are often referred to as conidia. Budding may occur within the ascus, but it is more common after the ascospore is discharged. In pure culture, budding may go on extensively, and thick-walled spores which probably represent a dormant stage of the fungus are found in old cultures. Thin- or thick-walled spores germinate by thin germ tubes. The young hyphae become binucleate early in pure culture and in the mycelium in the host. The nuclei continue to divide conjugately in the mycelium in host tissue. This is regarded by Martin (5) as a dicaryophase consisting of paired haploid nuclei. It comprises the largest portion of the life cycle of the organism.

Disease Cycle. There have been many different opinions among investigators concerning the method by which the fungus survives from season to season. Perennial mycelium in the twigs has been claimed by some. Overwintering spores have been suggested by others. Mix (6, 7) found no evidence of perennial mycelium and concluded that the fungus carries over as conidia on all surfaces of the dormant tree. Fitzpatrick (1) also concluded that conidia were the means of overwintering.

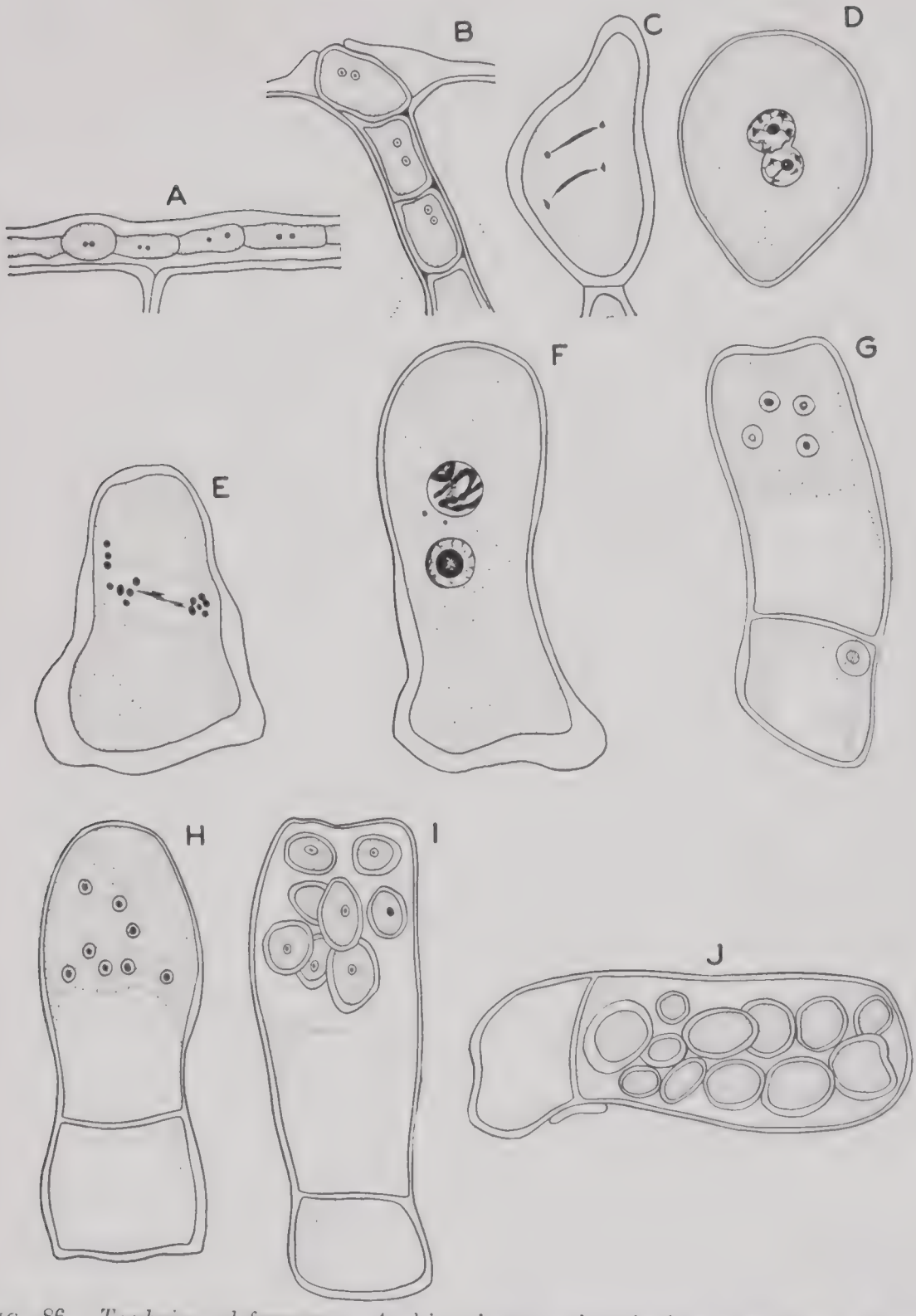

FIG. 86. *Taphrina deformans*. *A*, binucleate subcuticular mycelium; *B*, young ascogenous cell breaking cuticle; *C*, conjugate division in vegetative cell; *D*, fusion of nuclei in ascogenous cell; *E*, division of fusion nucleus at anaphase, eight chromosomes passing to each pole; *F*, elongating ascogenous cell containing two nuclei; *G*, four-nucleate ascus; nucleus still present in basal cell; *H*, eight-nucleate ascus with empty basal cell; *I*, typical ascus containing eight primary ascospores; *J*, ascus containing secondary spores. (*After Martin.*)

Germ tubes from conidia penetrate the young leaf directly into the cuticle. All organs become resistant as they grow older. The fungus makes its initial growth within the cuticular layer and then sends hyphae between the epidermal cells to the underlying parenchyma, where they become established as intercellular mycelium (1). From the mycelium in the mesophyll, branches penetrate between the cells of the upper epidermis (occasionally the lower epidermis as well) and ramify in a subcuticular layer. The subcuticular hyphae are composed of short binucleate cells, which round off as their walls thicken, thus forming between the epidermal wall and the cuticle a compact layer of chlamydospores (ascogenous cells). From the latter are formed asci which push through the cuticle.

It has long been established from observational evidence that the disease is most prevalent in areas where cool, moist spring weather prevails. Martin (4) found that the fungus produced conidia most profusely in pure culture at 12 to 16°C. Fitzpatrick (2) found that penetration occurred readily between 10 and 21°. However, a greater incidence of infection occurred at the lower than at the higher temperature. He attributed this difference largely to the greater rate of leaf maturation at the warmer level and suggested that the greater prevalence of the disease in cool wet weather was due primarily to the retarded growth of the host. Yarwood (11) described a diurnal cycle of ascus maturation. Elongation of the ascus begins in late afternoon and early evening; the ascus reaches the eight-nucleate stage about 5 A.M.; the ascospores appear mature about 8 A.M. The ascospores are discharged in greatest numbers the following evening about 8 P.M.

Control. This disease is controlled satisfactorily by a single fungicidal spray applied before the bud scales open. A dormant scalicide application of 5–50 lime-sulfur is effective, while, for leaf curl only, 1–20 lime-sulfur or 3–4–50 Bordeaux mixture is adequate. The application may be made in the fall or in the early spring. The comparative effectiveness of the two schedules may vary in different regions (3, 8–10).

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THE POWDERY MILDEWS

The powdery mildews comprise a large group of diseases which affect many host plants throughout the temperate zones. The pathogens belong to the family Erysiphaceae, all of which are obligate parasites. In all but a few forms the fungi produce a white to grayish growth of mycelium, conidiophores, conidia, and dark-colored cleistothecia on the surface of the host. They invade the latter only by haustoria, which in most species are confined to the epidermal cells. Conidia are borne in chains and are readily detached and dispersed by air currents. Many of the diseases are most severe under dry climatic conditions. The powdery mildew of cereals, on the contrary, is found to be most destructive in areas where moist conditions are more common.

The cleistothecia occur without any stroma and contain relatively few asci, in some species a single ascus being the rule. On the outer wall of the fruiting body conspicuous hyphal appendages occur; their morphology is a major criterion in distinguishing between genera. A key to the genera on the basis of appendages and number of asci follows:

Appendages hooked or coiled at the apex.	*Uncinula*
Appendages spike-like with a bulbous base.	*Phyllactinia*
Appendages dichotomously branched at the tip.	
Cleistothecium containing a single ascus.	*Podosphaera*
Cleistothecium containing several asci.	*Microsphaera*
Appendages simple or irregularly branched.	
Cleistothecium containing a single ascus.	*Sphaerotheca*
Cleistothecium containing several asci.	*Erysiphe*

Among the more common powdery-mildew diseases are those of grape, *Uncinula necator* (Schw.) Burr.; of apple, *Podosphaera leucotricha* (Ell. & Ev.) Salm.; of hop, rose, etc., *Sphaerotheca humuli* (DC.) Burr.; of gooseberry, *S. mors-uvae* (Schw.) Berk. & Curt.; of lilac, etc., *Microsphaera alni* (Wallr.) Salm.; of oak, chestnut, etc., *Phyllactinia corylea* (Pers.) Karst.; Karst.; of legumes, crucifers, etc., *Erysiphe polygoni* DC.; of cucurbits, etc., *E. cichoracearum* DC.; of cereals and grasses, *E. graminis* DC.

POWDERY MILDEW OF CEREALS AND GRASSES

Powdery mildew of cereals is important in the regions where humid conditions prevail during the growing season. It is most destructive on barley and wheat but occurs on other cereals and grasses. In the United States it is most severe in Pacific coast regions, in the vicinity of the Great Lakes, and in the Atlantic seaboard states. It is less serious in the cereal-growing region west of the Mississippi river and east of the Sierra Nevada and Cascade ranges. In Canada it is most important in British Columbia and in the five eastern provinces.

FIG. 87. Powdery mildew of barley. *A*, susceptible variety. Note abundance of conidia and cleistothecia. *B–D*, leaves from resistant varieties showing various degrees of necrotic reaction. (*After Dickson.*)

Symptoms. The disease appears in the form of white, later gray-tan, mildewy areas on aerial parts of the plants; these may enlarge and coalesce until most of the plant surface is involved. Conidia may accumulate until they form a visible, dusty, powder, part of which may be removed by agitating the plant parts. Cleistothecia may or may not form. When they occur, they are seen, usually after the disease has prevailed for some time, as macroscopically visible black dots scattered singly over the mycelial mat and not attached directly to the host (Fig. 87).

Infection results in temporary stimulation of respiratory activity of the tissue and in chlorosis beneath and around the fungus. There is also an increase in transpiration of the host and further water loss due to the transpiration of the fungus. There follows a gradual reduction in thriftiness of the host plant, which is reflected in reduced yield and in some reduction in normal plumpness of the kernel.

After chlorosis of the tissue beneath the fungus mycelium has occurred, the reproductive activity of the mycelium declines, and sporulation is more profuse around the advancing edge of the lesion. Chlorophyll may then reappear beneath the center, giving rise to conspicuous green spots.

Apparently this chlorophyll does not become active in photosynthesis, however (1, 2).

The Causal Organism. *Erysiphe graminis* DC. The mycelium is branched, hyaline, and uninucleate. The conidia are borne in chains on short conidiophores arising from the mycelium at a right angle with the host surface (Fig. 88). They are mostly uninucleate, occasionally binucleate, hyaline, oblong to ovate, 8 to 10 by 25 to 40 μ, and they germinate by a simple germ tube. Cherewick (10) secured perithecial development in single-conidium lines, showing that at least some races of the fungus are homothallic. When ascospores from a field where conidia yielded only races 4 and 6 of *E. graminis tritici* were pure-lined, races 4, 6, 9, and 10 were found, indicating that cleistothecia may arise from the fusion of thalli of distinct genetic composition. While the nuclear history in the development of the sexual stage has not been studied for *E. graminis*, it has been worked out for some other species. The ascogonium and antheridium arise usually on neighboring hyphae and fuse, and an antheridial

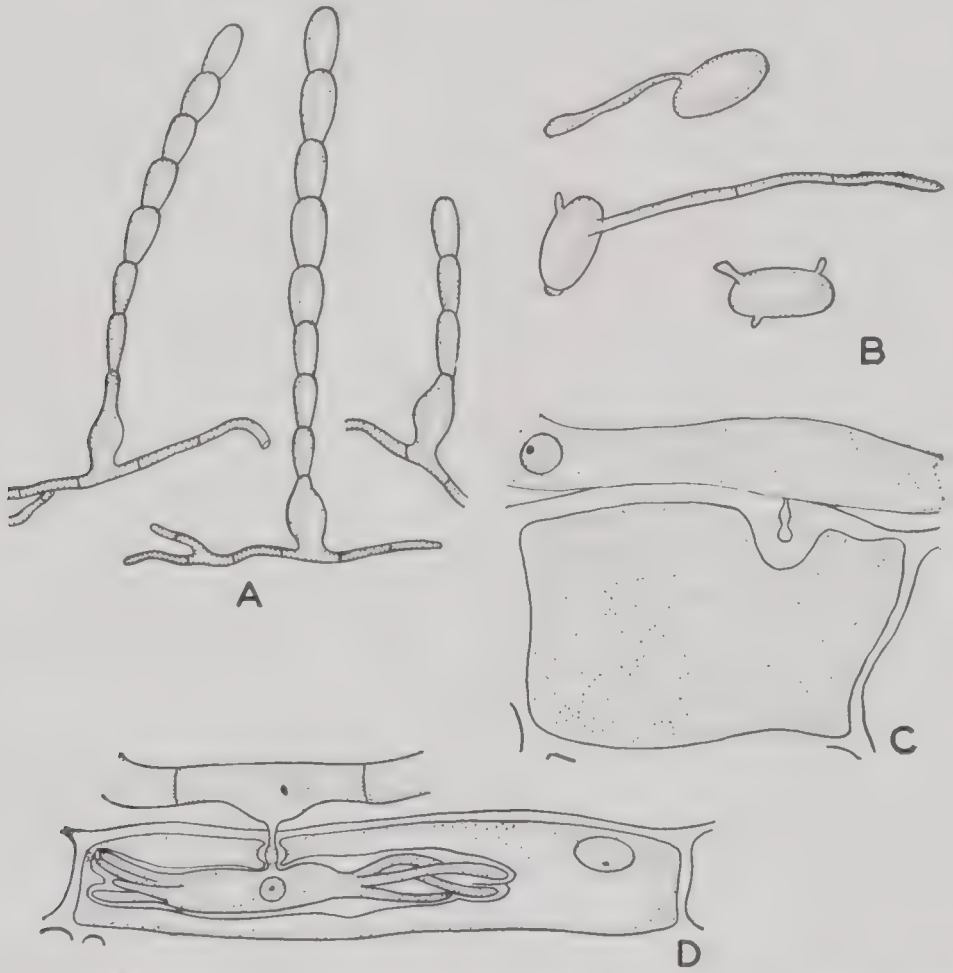

FIG. 88. *Erysiphe graminis*. *A*, conidiophores and conidia; *B*, germinating conidia; *C*, penetration of cuticle and epidermal wall; *D*, haustorium. (*A*, *B after Reed*; *C*, *D after G. Smith.*)

nucleus enters the ascogonium, from which ascogenous cells arise. In some species the male nucleus degenerates in the antheridium, and the ascogonium develops parthenogenetically to form asci. Harper (16) described two nuclear fusions, one in the ascogonium and one in the ascus.

The cleistothecium is about 200 μ in diameter, with simple appendages, containing usually eight, sometimes four, ascospores (10 to 13 by 20 to 23 μ), which are hyaline, continuous, and uninucleate.

Disease Cycle. In general the fungus overwinters as a mycelial mat on leaves of grasses and autumn-sown grains. The perithecia appear to be of secondary importance in overwintering and as a source of inoculum in the spring. Conidia are produced most abundantly in relatively cool moist environment. They are dispersed readily by air currents and are the chief secondary inoculum during the usual cereal-growing season. They are not long-lived, however. The conidia germinate over a range of 0 to 35°C. with an optimum at 10° (10). Clayton (11) reported that they require a relative humidity of 95 per cent or higher for germination. Cherewick (10), on the contrary, reported germination of conidia at zero humidity. Upon germination the germ tube forms an appressorium in juxtaposition with the host cuticle. A hyphal peg penetrates the cuticle and the epidermal cell. The development of haustoria in the host cell varies with the degree of resistance or susceptibility of the host. In a susceptible variety the haustorium is large in proportion to the cell and has several finger-like projections at each end. Haustoria also develop in resistant varieties, but their size is roughly in inverse proportion to the degree of resistance, they being reduced to small spheroid lobes in some cases. In resistant varieties in which necrotic spots appear as a result of infection, haustoria develop as usual, the extent depending again on the variety concerned, and the cell protoplasm collapses later.

Infection and disease development are best at 15 to 20°C. Temperatures alternating from about 10 to 20° are most favorable for spread of the pathogen and for development of the cleistothecia. Alternate drying and wetting are essential for maturation of the ascospores. As noted by Yarwood (37) for several species of *Erysiphe*, Cherewick (10) found that infection was reduced by sprinkling plants with water. Yarwood (37) attributed this to injury to the conidiophores which prevented sporulation. It is still not fully explained why most species of the powdery mildews are abundant and destructive in semiarid regions or during dry spells in humid regions, while *E. graminis* is most epidemic in cool, moist, or foggy climate. Tapke (32) suggested that the growing conditions of the host previous to exposure to the fungus may affect the extent of infection. Conditions promoting succulence and probably relatively thin cuticle are more favorable to infection. Numerous observations have

been recorded to indicate that variation in nutrient may affect susceptibility to mildew (30, 34, 35).

Varietal Resistance and Pathogenic Specialization. The development of varieties resistant to mildew has received much attention, particularly with reference to barley and wheat. This problem is complicated by the fact that this fungus, as well as other members of the Erysiphaceae which have been studied critically in this regard, is made up of forms and races that are specialized to genera, species, and varieties of the host plants. The existence of distinct physiologic forms of *E. graminis* was first shown by Marchal (24) in France in 1902. He distinguished seven forms on the basis of the principal host genera which were affected, *i.e.*, *E. graminis tritici*, *hordei*, *avenae*, *secalis*, *poae*, and *agropyri*. Physiological races within *E. graminis hordei* were first described by Mains and Dietz (22) in 1930 and within *E. graminis tritici* by Mains (20) in 1933. Mains and Dietz defined races 1 to 5 of *E. graminis hordei* on the basis of reaction of barley varieties tested. Tidd (33) described races 6 and 7 and the discovery of several new races in Canada has increased the number to 12 (25). Honecker (17, 18) in Germany, using a different list of differential varieties, described nine races. Mains (20) originally described two races of *E. graminis tritici;* the number has been increased to five (25) in the United States and Canada, while Schlichtling (29) in Germany described six races distinct from race 1 and 2 of Mains. It is to be expected that other races will be distinguished as more collections of the fungi and more varieties of the host plants are studied.

Varieties of wheat and barley differ widely in their reaction to one or another race within the respective form of *E. graminis* to which they are susceptible. Mains and Martini (23) catalogued the reaction of a long list of barley varieties with reference to races 1, 2, and 3. Honecker (18) has made a similar survey in Germany. Varietal-resistance studies on both wheat and barley have been reported from various countries. Varieties which are resistant to races prevalent in a given locality may eventually succumb to the disease as other races appear. The process of improvement of barley and wheat for resistance to mildew, for resistance to the important rust diseases and for desirable agronomic qualities, is thus a continuous one. Caldwell and Compton (9) have reported progress in this direction in the soft-winter-wheat area of the Mississippi valley. Newton and Cherewick (25) have summarized the progress on wheat and barley in Canada.

Briggs and associates (3, 8, 13, 31) have studied the genetic basis of resistance in barley to race 3 of *E. graminis hordei*. Hanna, Goldfoil, Algerian, Kwan, and an unnamed variety, S.P.I. 45492, were shown to differ from susceptible Atlas by one dominant factor. Crosses within the group of

five resistant varieties, however, showed that factors in the first four were different and that those in Algerian and S.P.I. 45492 were the same. The genes have been designated as Ml_h, Ml_g, Ml_a, and Ml_k; Ml_a and Ml_k are linked. Arlington Awnless, Chinerme, and Nigrate were found to have two dominant factors, each differing from the first four. These last three varieties were found to have at least one factor in common. The two newly described genes were designated as Ml_x and Ml_y. Later the gene Ml_x was found in Psaknon, and the designation was changed to Ml_p. Another factor which is recessive in effect was shown to occur in Duplex along with Ml_h and Ml_p. It was designated as ml_d. Favret (13) described a dominant gene Ml_m in Monte Cristo and Engledow India, and a recessive gene ml_w in West China variety. Thus nine distinct genes, located at distinct loci, have been shown to control resistance to race 3. Tidd (33) demonstrated in Nepal, a variety susceptible to race 3, a gene dominant for resistance to race 6. Dietz (12) described, in several varieties, resistance to race 4, which behaves as a single recessive factor.

It is of particular importance to note that a number of distinct genes in barley control independently resistance to one race of *E. graminis hordei*. This fact lends strength to the possibility that in the numerous agronomic varieties which may be assembled, other genes may be found which, through breeding procedures, may eventually be combined in suitable commercial varieties. Complete control of the disease through resistance may thus be accomplished.

Control. The control of powdery mildew resolves itself at present into avoiding varieties which are known to be extremely susceptible in the locality concerned and into taking advantage of any of those which are otherwise satisfactory and carry resistance to some of the prevalent races.

The disease may be controlled by dusting with sulfur, which acts both as an eradicant and as a protectant. Except in special cases, however, the return from the application of this measure has not been considered sufficient to warrant its use.

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DUTCH ELM DISEASE

The Dutch elm disease acquires its name from the fact that it was first described in Holland (1921), where it rapidly decimated elm plantings. There is no proof that the pathogen originated in that country, and it may well have been imported from another one. It was recorded in relatively rapid succession in many parts of Europe and Asia. It was first found in the United States in Ohio in 1931 and in Canada in the province of Quebec in 1944. The details of its incidence and spread and of the attempts at restriction and eradication in the United States are related in Chap. 15. A summary of the occurrence of the disease in Europe up to 1936 is given by Clinton and McCormick (2). A large number of species of *Ulmus* are hosts.

Symptoms. The effect of the disease appears first on the leaves. Various degrees of yellowing, curling, browning, wilting, and defoliation occur. The progress of symptoms is more rapid on succulent, rapidly growing trees than on slowly growing or senile ones. The xylem becomes brownish in color, and brown gummy materials accumulate. The symptoms are usually not sufficiently distinctive to permit certain diagnosis of the disease without laboratory study.

The Causal Organism. *Ceratostomella ulmi* Buisman, 1932.

Synonymy: *Ophiostoma ulmi* (Buisman) Nannfeldt, 1934.
Conidial stage: *Graphium ulmi* Schwarz, 1922.

The mycelium is white in culture, turning yellowish en masse with age and becoming covered with conidia which give it a yellowish shiny surface. Conidia are borne at the tips of short, simple or branched, hyphae, and they may bud rapidly. They are hyaline, elliptical, 2 to 3.5 by 4.7 to 10 μ. Coremia may form on the surface of cultures as brown to black hair-like projections above the colony. They may develop also on the bark of affected branches as erect dark stalks with a colorless flaring head, to which conidia adhere as a white to yellowish droplet (Fig. 89).

The fungus is heterothallic. The perithecia are spherical, black, single or in groups (105 to 135 μ), with a neck, varying from 267 to 450 μ in length, having a ciliate fringe at the ostiole.

The asci disintegrate early, leaving the hyaline, continuous, slightly curved ascospores (1.5 by 4.5 to 6 μ) in the perithecial cavity, from which they are discharged through the neck canal to accumulate in a droplet at the ostiole.

Walter (16) found the pathogen to contain a wide range of cultural races. Tyler and Parker (12) observed that variants arising from monoconidial lines had various degrees of pathogenicity and maintained their respective characteristics through 7 years of culturing on artificial media.

Disease Cycle. The pathogen is one of the Ascomycetes which does not require the ascigerous stage to complete the disease cycle and produce epidemics. In the United States perithecia are not often found, but they do occur in nature in Europe. Although the conidia may be disseminated by water and to a very limited extent by air currents, they are largely dependent upon an insect vector for dissemination and infection. The spores survive in dead or infected living trees largely in the tunnels made by boring insects. A number of species of insects have been shown to be vectors, but two are the most common ones in the United States. These are the introduced smaller European bark beetle (*Scolytus multistriatus* Marsh.) and the native elm bark beetle (*Hylurgopinus rufipes* Eich.). The spores of the fungus are carried primarily on the exterior of the bodies of beetles. Dead logs of elm or other trees may be infected, and a male beetle may contaminate numerous females during mating. The fungus is known to remain viable on the bodies of insects for 3 months at 10 to 20°C. and for 2 years or more on beetles kept frozen at −5°C. (5).

The fungus grows on culture media within the range of 3 to 33°C. with an optimum at 27°. Conidia germinate from 12 to 33° with an optimum at 27°. At 18° and lower, coremiospores tend to bud. Wilt appears within 5 days after inoculation at 26 to 29°, which is the optimum. The disease develops over a range from 15 to 29°, but daily exposure of inoculated trees which have completed terminal growth to a temperature of 32 to 37° completely inhibits the disease.

Infection takes place readily through wounds in wood, leaf, midrib, and succulent shoots, but not through the unwounded surface except that of freshly exposed wood. High humidity favors infection when inoculation

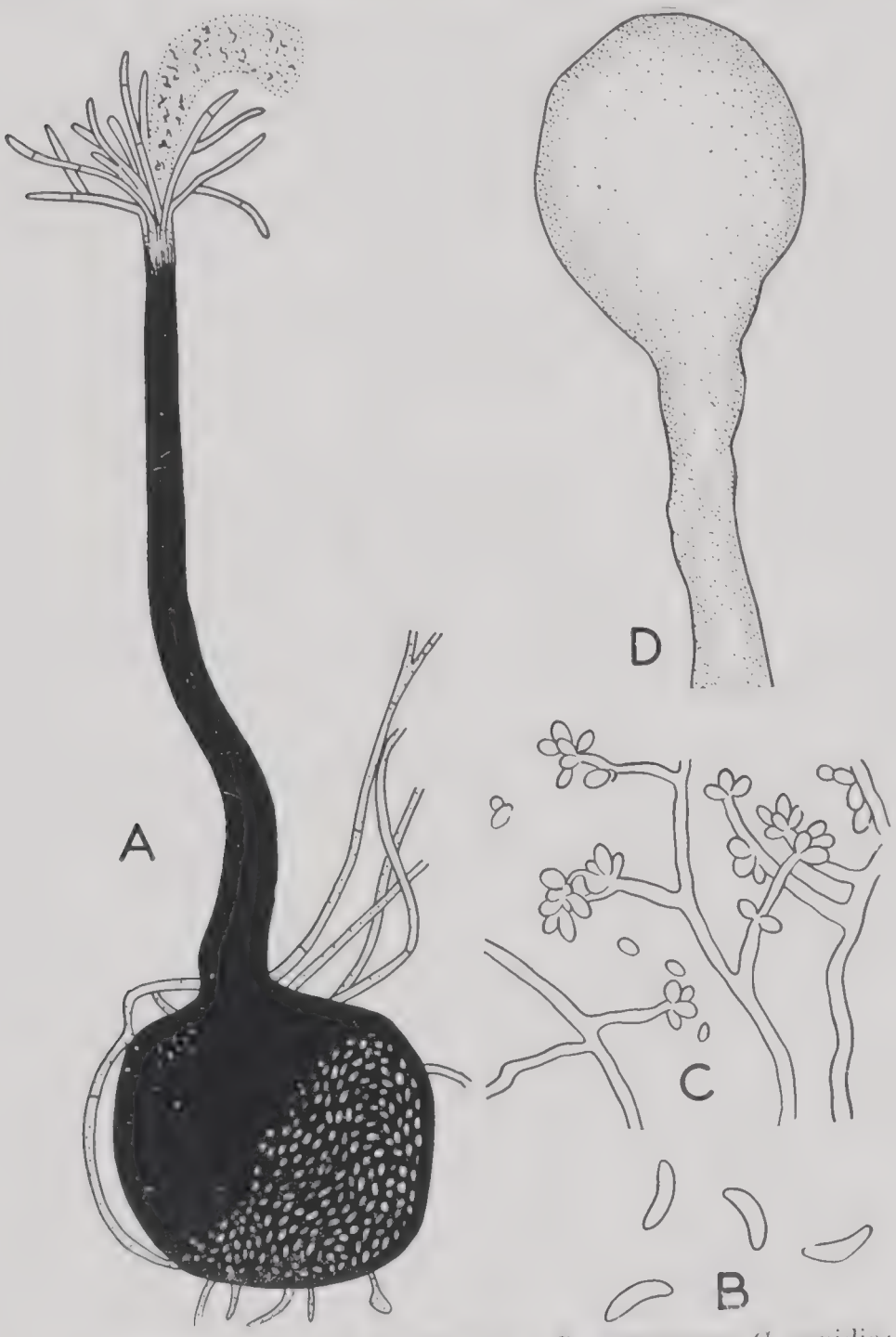

FIG. 89. *Ceratostomella ulmi*. *A*, perithecium; *B*, ascospores; *C*, conidia; *D*, mass of viscid spores at tip of coremium. (*A*, *B after Buisman; C*, *D after Clinton and McCormick.*)

is made through artificial wounds. The fungus does not survive in the soil, but transmission of the pathogen from tree to tree through natural root grafts may occur. Banfield (1) showed that conidia were distributed

rapidly in the transpiration stream. In June, spores were recovered 30 ft. above the point of injection within 3 hr.

In England, Walter (17) reported that coremia and perithecia occurred in abundance under humid conditions in dead, dying, and fallen elm trees and logs. They occurred in galleries of bark beetles, on the inner bark surface, between inner bark flakes, on the outer xylem surface, and on cut or broken surfaces. Development of perithecia ceased during the period from November to April.

Varietal Resistance. Soon after the appearance of the disease in Holland a study of the relative resistance of species of *Ulmus* was started. The results through 1938 are summarized by Went (19). The expression of inherent resistance in seedlings is influenced by the prevailing environment and the vigor of the tree. In general, the disease progresses more rapidly in vigorous than in slowly growing trees. The most resistant species are to be found among the Asiatic forms, of which *U. pumila* L. is one of the least susceptible. In Italy this species has promise for replacing the susceptible *U. foliacea* Gilib., which is used as a vine support, but in Holland it is too susceptible to *Nectria cinnabarina* Fr. to be of commercial value. The American species are all very susceptible. The European species were all too susceptible to replace the common one used (*U. hollandica* Mill.), but within *U. foliacea* a small number of seedlings were found which exhibited various degrees of resistance. One of these (No. 24) was superior, and the clone therefrom, under the name Christine Buisman elm, has now been planted extensively. Another seedling of the same species (No. 62) was ready for distribution to nurserymen in 1946. Hybrids from crosses between resistant clones and desirable susceptible species offer promise (20). Breeding programs are under way in other European countries and in the United States.

Control. Until suitable resistant elms for various regions and purposes, are secured by breeding methods, no entirely satisfactory control measures are available. Restriction of transport of elm trees and elm wood from infested areas will reduce the chances of introduction of the pathogen into new locations. Local spread can be reduced by prompt removal of dead limbs and disposal of all deadwood before May in northern latitudes, since such material is the center of overwintering beetles. Deadwood should be burned or sprayed with a suitable insecticide (18).

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MYCOSPHAERELLA AND *ASCOCHYTA* DISEASES OF PEA

There are three closely related diseases of garden pea (*Pisum sativum* L.) incited by as many species of *Ascochyta*. One of these pathogens has a perfect stage which belongs to the genus *Mycosphaerella*, and the disease it incites is referred to as *Mycosphaerella* blight. Another species (*A. pisi* Lib.) incites *Ascochyta* leaf and pod spot, while a third (*A. pinodella* L. K. Jones) incites *Ascochyta* foot rot. While one or the other of these

diseases has been known for centuries in Europe, the first technical description of any of the organisms was that of Libert, who described *A. pisi* in 1830. The next species, *Mycosphaerella pinodes*, was described by Berkeley and Bloxam in 1841 as *Sphaeria pinodes*. The third species (*A. pinodella*) was described by L. K. Jones in 1927.

The diseases are world-wide in occurrence. In the United States *Mycosphaerella* blight and *Ascochyta* foot root are the most important.

FIG. 90. *Mycosphaerella* blight on leaf, blossom, and pods of garden pea. (*After Hare and Walker.*)

Mycosphaerella blight is most severe in pea-growing areas east of the Continental Divide and in Pacific coast areas where peas are grown during the rainy season. It is less common in semiarid irrigated areas of the western intermountain region. Foot rot has the same distribution but is more important than blight under irrigation. Blight is important in southeastern states, where field peas are grown for winter forage or as a cover crop.

Symptoms. *Mycosphaerella Blight.* Small purple spots appear on the leaves (Fig. 90). They remain small without definite margins, or they

enlarge up to 6 mm. in diameter, turning brown to black, assuming definite margins and often a zonate appearance. Affected leaves may be killed, drying out, clinging to the stem, and giving the lower portion of the plant an appearance suggestive of cured legume hay (Fig. 91). Stem lesions are similar in color but are elongate (up to 1 cm.), often extending upward and downward from the point of attachment of an infected leaf petiole.

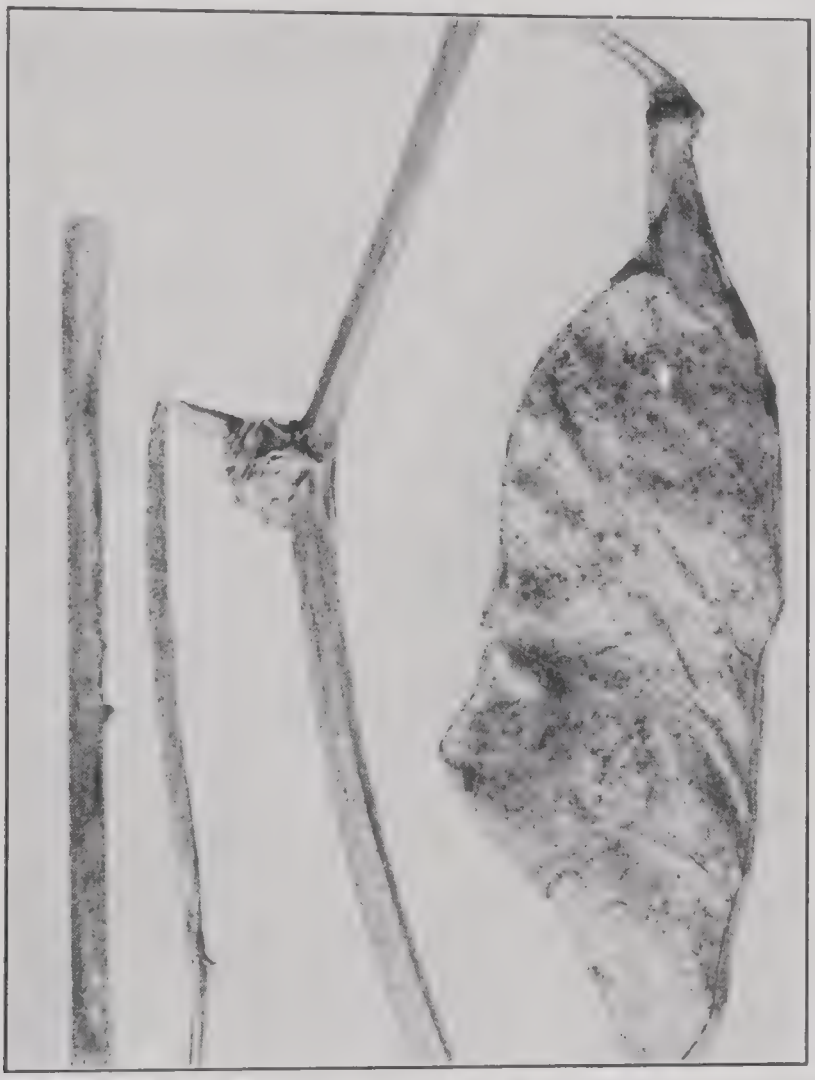

FIG. 91. Perithecia of *Mycosphaerella pinodes* on dead stems and pod of pea. (*After Hare and Walker.*)

Stem lesions may coalesce to give the entire lower stem a blue-black cast. When small pin-point lesions appear on the various flower parts, blossom drop commonly follows. When environment is very favorable, heavy infection on immature pods may cause shrinkage of the pod wall and impair market quality. Infected seed may show no symptoms, or various degrees of shrinkage and discoloration may be visible. Infection from diseased seeds results in a foot rot, starting at the point of seed attachment and advancing up the stem and down the taproot. The stem lesion may extend to a point above the soil line, and young plants may be killed.

Ascochyta Foot Rot. The symptoms on stem and foliage are not unlike those of blight, but they are seldom very abundant. Foot-rot lesions are the predominant ones with this disease.

Ascochyta Blight and Pod Spot. The lesions on leaves are tan-colored, with dark brown margins; on stems they are similar but definitely sunken. On pods they are circular, sunken, tan-colored with dark margins. Black pin-point-size pycnidia are often conspicuous. Basal stem and taproot lesions are rare or inconspicuous.

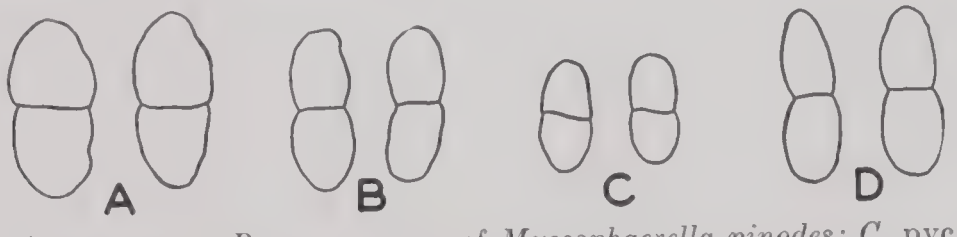

FIG. 92. *A*, ascospores; *B*, pycnospores of *Mycosphaerella pinodes; C*, pycnospores of *Ascochyta pinodella; D*, pycnospores of *A. pisi*. (*After Hare and Walker.*)

The Causal Organisms.

1. *Mycosphaerella pinodes* (Berk. & Blox.) Stone, 1912.

SYNONYMY:

Conidial stage: *Ascochyta pinodes* (Berk. & Blox.) Jones, 1927.

Ascigerous stage:

Sphaeria pinodes Berk. & Blox., 1841.

Sphaerella pinodes (Berk. & Blox.) Niessl.

2. *Ascochyta pisi* Lib., 1830.
3. *Ascochyta pinodella* L. K. Jones, 1927.

All three species produce pycnidia on the host and in pure culture. The pycnospores are all hyaline and typically two-celled. Hare and Walker (2) report average dimensions of pycnospores from potato-dextrose agar cultures for the three species as follows: *M. pinodes*, 4.5 by 12.3 μ; *A. pinodella*, 3.7 by 8.0 μ; *A. pisi*, 4.2 by 13.9 μ. The pycnospores of *M. pinodes* and *A. pinodella* are much alike in shape, differing chiefly in size; those of *A. pisi* differ from those of *M. pinodes* in shape, being slightly narrower and longer (Fig. 92). The three species have distinctive types of growth on potato-dextrose and oat agar (2, 3).

M. pinodes is homothallic. The perithecium is globose with a beak-like ostiole. Asci are cylindrical-clavate with a wall made up of two membranes. The inner membrane is thickened at the apex and is provided with an apical pore. Upon ejection of ascospores the outer membrane ruptures at the tip, and the inner membrane stretches to approximately three times its normal length. The spores move toward the apex, and when the membrane ruptures at the pore, the spores are ejected, and

the stretched membrane contracts (6). Ascospores are hyaline, two-celled, 6 to 10 by 15 to 10 μ (Fig. 92).

Disease Cycle. All three pathogens are carried in infected seed, and they also overwinter in infected plant debris. In the case of seed-borne mycelium with *M. pinodes* and *A. pinodella*, infection begins at the point of seed attachment, and foot-rot lesions develop, often killing young plants, from which secondary spread may occur through dissemination of pycnospores. In the case of *A. pisi*, primary lesions appear on the first leaves. Primary infection by each species may also arise from pycnospore inoculum in infected debris. In the case of *M. pinodes* the development of perithecia in infected plant parts of the previous season constitutes an important part of the disease cycle (Fig. 91). In pure culture, perithecia develop most rapidly and abundantly at 16°C.; few occur at 24° and above; they are as abundant at 4°, 8°, and 12° as at 16° but are slower to develop at the three lowest temperatures (2). In a study of the disease cycle in Wisconsin (2), it was found that the mature pycnidia and pycnospores which overwintered on pea straw out of doors were no longer viable in the spring, but new fruiting bodies of both types developed promptly, furnishing inoculum for primary infection. Pycnospores depend upon water for discharge and dissemination. The perithecia require a longer period for maturation and usually begin to discharge ascospores early in June. When mature perithecia absorb water, ascospores are discharged through the ostiole for a distance of 2 to 3 mm., whence they are carried by air currents for $^1/_4$ mile or more. They are responsible for much wider dissemination than that effected by pycnospores.

Pycnidia and perithecia are produced throughout the growing season and after harvest on pea stubble and volunteer seedlings. On the host plants perithecia form over a wider range of temperature than in culture, and temperature has no obvious effect on extent and rate of discharge. The most important environal factor during the growing season, then, is moisture; it is required for spore discharge and for infection.

In view of the fact that rainfall is very light in irrigated regions of the western states, the spread of *Mycosphaerella* is greatly reduced. This is an important factor in seed production. Most of the pea seed was produced in eastern United States before 1915, but about that date a general shift to western states took place. This was followed by a general reduction in severity of *Mycosphaerella* blight because of reduction of seed-borne inoculum. In general, this situation still holds where crop rotation is adequate to eliminate the fungus in overwintering debris. A study of disease incidence in relation to number of years between pea crops was made in Wisconsin in 1942 (7). When early-sown peas were considered, there was a definite inverse correlation between incidence of disease and

length of the pea-free period. When late-sown crops were considered, the correlation did not hold. This was considered to be due to wind-borne inoculum from distant or adjoining fields, a factor which did not enter in in time to affect early-sown fields.

Control. There are two major approaches to control. The first of these is elimination of seed-borne inoculum. This can be accomplished to a large extent by production of seed in areas of low rainfall during the growing season. The second approach is the elimination of the organism in infected overwintering plant parts. The fungi are not soil inhabitants and do not survive after the plant material is thoroughly decomposed. They can be largely eradicated by a 3- or 4-year rotation. In addition, it is necessary to select fields which are 80 rods or more distant from fields of the previous season in order to avoid wind-blown ascospores from infected stubble, parts of which may be on the soil surface in the next crop season. Refuse from pea strawstacks, where dry peas are grown, and from the outside of pea-vine silage stacks, where canning peas are grown, is a potential source of inoculum and should be disposed of properly. While treatment of pea seed with protectants is of value for control of pre-emergence damping-off, it is of little or no value in controlling seed-borne inoculum of the three *Ascochyta* pathogens.

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APPLE SCAB

Apple scab is world-wide in distribution, occurring in most areas where the host is grown. Only in very arid regions or in those where relatively high average temperatures prevail is it absent or of little importance. It is most destructive in relatively cool, humid climate. In the north

central and northeastern United States and in northwestern Europe it is a limiting factor in apple production unless a rigid program of protective control measures is followed.

The disease was first reported in Sweden in 1819 by Fries. It was noted in Germany in 1833; in the United States in 1834; in England in 1845; in Australia in 1862. Apple and crabapple are the only economically important hosts. Several other species of *Malus* are susceptible to the pathogen.

Symptoms. The most conspicuous and important phases of the disease are on leaves and fruits. Lesions occur on both sides of the leaves, usually developing earliest on the lower side. They appear in the form of olivaceous spots slightly darker than the leaf lamina, turning dark brown to black and assuming a more or less velvety appearance with age. Two general types of lesions occur. Those on the upper side of the leaf usually are the more conspicuous and have rather definite margins. Most of those on the lower side merge more or less into the healthy tissue without a conspicuous or definite margin and tend to extend along vein and midrib. There may be slight distortion of the leaf surface, the lesion forming a convex surface with corresponding concave area on the opposite side. Symptoms on the calyx lobe are quite similar to those on leaves. On the remainder of the fruit dark, almost black, small spots appear which enlarge much more slowly than on leaves. As the growing spot becomes older, the center loses the velvety appearance and becomes brown and corky. The loosened cuticle of the fruit appears as a whitish band around the spot (Fig. 93). When fruits are infected in an immature stage, distortion of growth follows, and cracking of the surface covered by the lesion may occur. The appearance on twigs, which is uncommon in most parts of the United States, consists of blistering and rupturing of the bark, giving them a scurfy appearance.

The Causal Organism. *Venturia inaequalis* (Cke.) Wint., 1875.

Synonymy:

Conidial stage:

Spilocaea pomi Fr., 1819.
Cladosporium dendriticum Wallr., 1833.
Fusicladium dendriticum (Wallr.) Fckl., 1869.
Fusicladium pomi (Fr.) Lindau.

Ascigerous stage [see Shear (*26*)].

Sphaeria cinerascens Fckl., 1863.
Sphaerella cinerascens Fckl., 1865.
Sphaerella inaequalis Cke., 1866.
Didymosphaeria inaequalis Niessl., 1881.
Venturia inaequalis Aderh., 1897.

Endostigme inaequalis Syd., 1923.
Spilosticta inaequalis Petr., 1940.
Spilosticta inaequalis Petr., 1940.
Endostigme cinerascens Jørst., 1944.

The mycelium is hyaline when very young, becoming olivaceous rapidly and turning to a reddish brown with age. It is septate and branching, and the cells are uniformly uninucleate (2). Stromata, one to several layers thick, consisting of densely packed rounded mycelial cells, hyaline at first, darkening with age, form beneath the host cuticle. From the

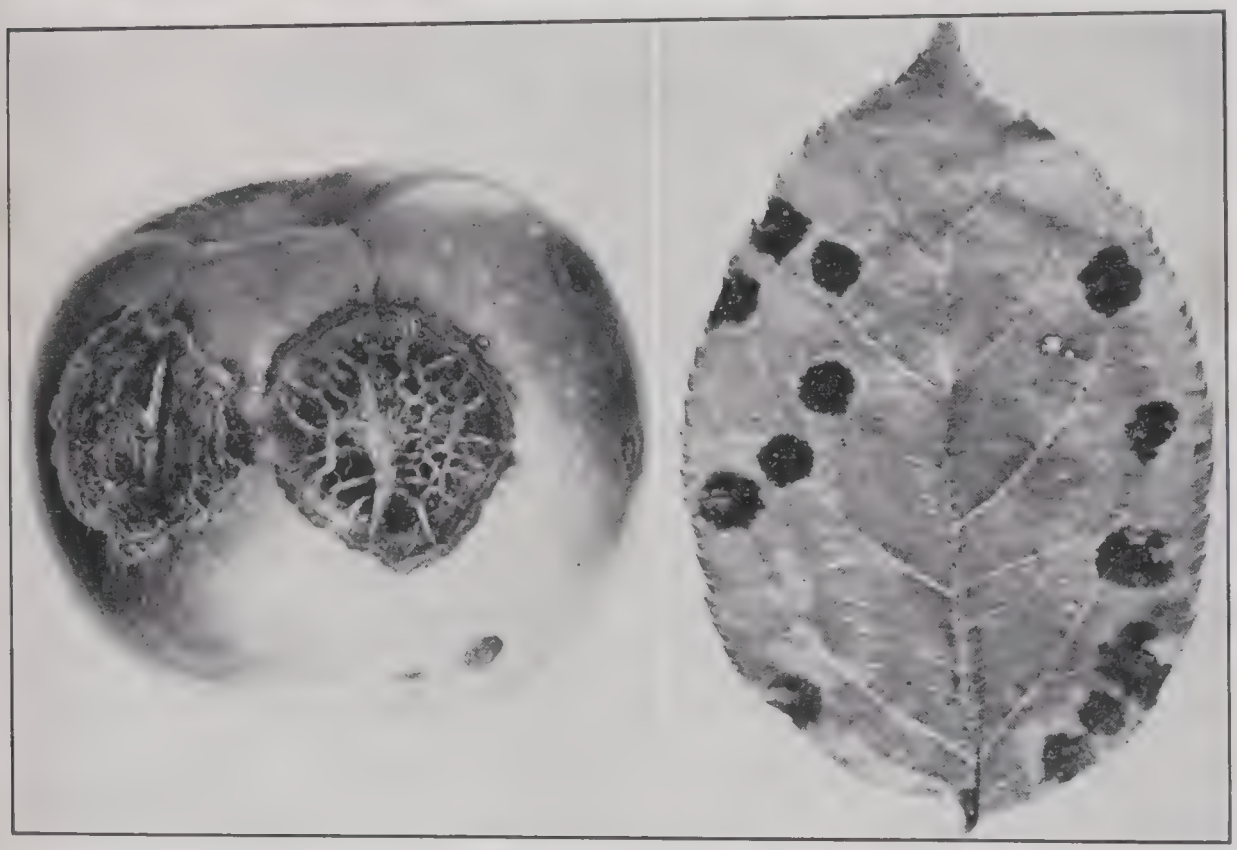

FIG. 93. Apple scab.

stroma arise short, reddish-brown conidiophores, which bear conidia at the apex (Fig. 94). The conidiophore grows on slightly at the sporiferous apex after each conidium is formed (1). An old conidiophore has a series of closely placed transverse septa near the tip, each representing the point at which a spore was formed. Conidia (6 to 9 by 12 to 22 μ) are hyaline at first, becoming rapidly reddish brown in color; they are variable in shape, continuous or one-septate. Cells of conidiophore and conidium are uninucleate.

Perithecia appear in dead leaves, maturing chiefly in early spring in northern regions (28). They are dark olive-green to brown, partially embedded in the tissue, spherical to subspherical, 90 to 160 μ in diameter,

slightly papillate at the ostiole, and the latter is surrounded by several setae 25 to 75 μ in length (Fig. 94). The perithecium is initiated by spiral growth beginning at the apex of a hypha and enclosing the latter. The perithecial wall arises from a portion of the fundament, while within

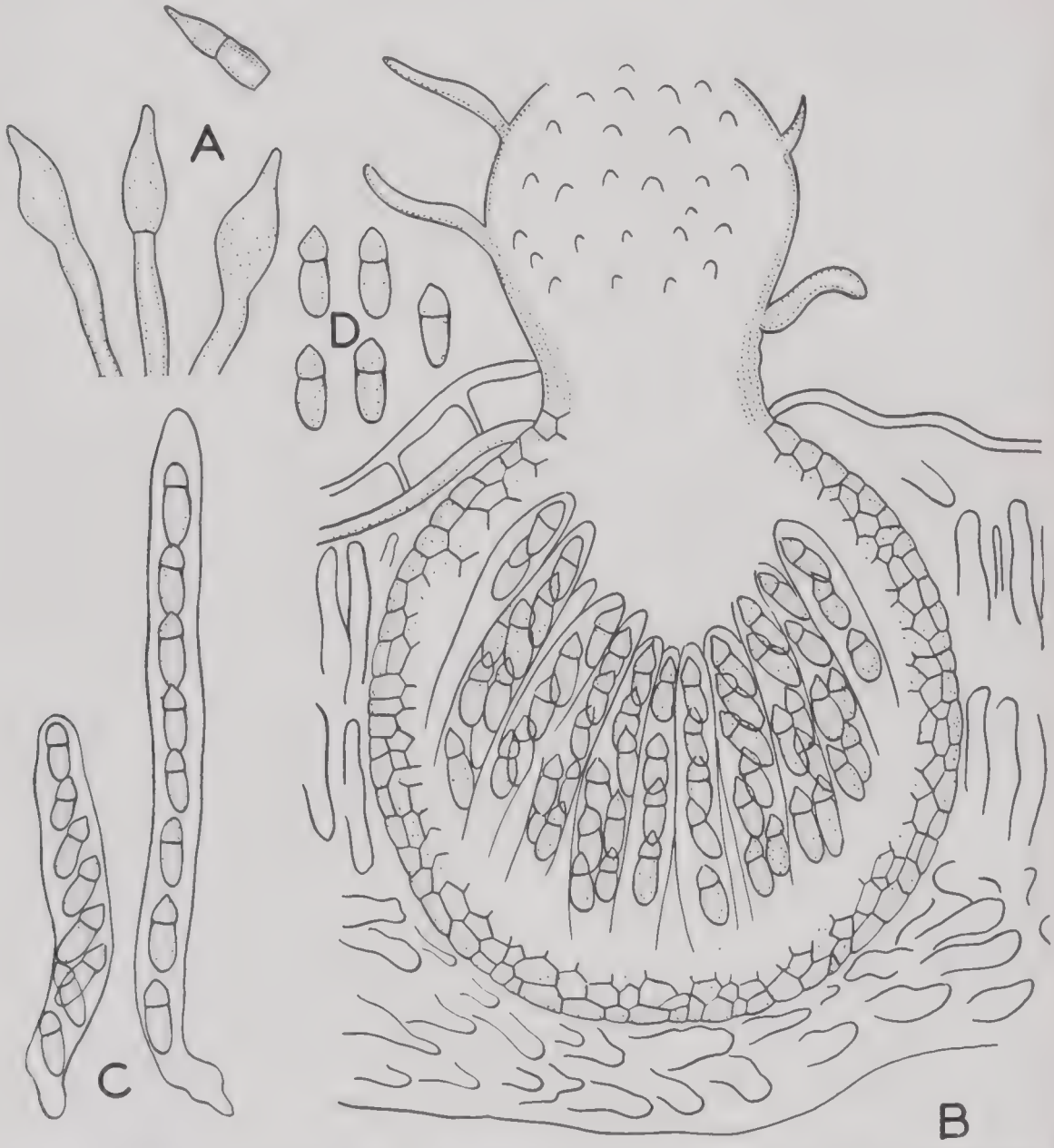

FIG. 94. *Venturia inaequalis*. *A*, conidiophore and conidia; *B*, perithecium; *C*, asci; *D*, ascospores. (*After Aderhold and Ruhland.*)

the latter the ascogonium initial develops, becomes curved and septate, and gives rise to a well-defined trichogyne. The antheridium arises from another hypha, becomes multinucleate, and attaches itself to a trichogyne. A pore in the adjoining walls forms; nuclei pass from the antheridium into the ascogonium, the septa of which have meanwhile dissolved (2). There is a mingling of nuclei from both organs. The ascogenous hyphae arise,

and crosiers form at their tips. A binucleate penultimate cell forms, and the two nuclei fuse promptly to form the diploid stage. The ascus elongates, and three successive divisions in the nuclei occur, the first of which is the reduction division. The cytoplasm is delimited around each of the eight nuclei, and the same number of uninucleate haploid ascospores is formed. The nucleus in each spore divides, and the formation of a septum results in a spore with two uninucleate cells of approximately equal size. The spores elongate rapidly, the cells growing at different rates, to form the characteristic mature ascospore with two cells of unequal size. The mature spores are olivaceous in color, 5 by 11 to 15 μ, and occupy the greater portion of the mature ascus, which is 6 to 12 by 55 to 75 μ in size.

Study of monoconidial isolates has shown this fungus to consist of numerous strains (biotypes) which differ in cultural characters and in pathogenicity (20). Keitt and associates have made fundamental contributions to the inheritance of pathogenicity through genetic studies of this organism (13, 15, 16, 17). The fungus is hermaphroditic, *i.e.*, any given ascospore line produces both ascogonia and antheridia. These haploid lines, however, are self-incompatible. When eight ascospore lines are derived from a single ascus, they fall into two groups. Those within each group are incompatible with one another (intragroup incompatible), but any line within one group is compatible with any one in the other group (intergroup compatible). By breeding procedure it was shown that one pair of genes controls pathogenicity to Haralson and Wealthy varieties, while another independent gene pair controls pathogenicity to Yellow Transparent and McIntosh.

Disease Cycle. The fungus overwinters as stromatoid mycelium in twigs on the tree and as mycelium and incipient ascocarps in fallen leaves. Infected twigs are not common except in certain very susceptible varieties and under special environal conditions, and they are not regarded as common sources of primary inoculum in the United States. After leaves fall, the saprophytic stage of the fungus begins (28). Mycelium from the indefinitely margined lesions and from the periphery of definitely margined lesions penetrates from the subcuticular stroma into the interior of the leaf. Perithecia are initiated during the fall and winter and mature in the spring to produce ascospores which form the chief inoculum for primary infection. The optimum temperature for initiation and early growth of perithecia is about 13°C. The optimum for maturation of ascospores is about 20°, while temperatures above 24° are detrimental if leaves are moist (11). Sufficient moisture in the leaves to make them fully pliable, and alternation of wetness and dryness, are conditions most favorable to maturation. Heavy ascospore discharges follow periods of rainfall over a period of 5 to 9 weeks in the spring in northeastern Wisconsin. In

general, even a heavy dew is insufficient to promote heavy discharge. Ascospores are ejected forcibly into the air and are then disseminated by wind.

The mixed buds containing the floral primordia are first to open in the apple, exposing the tips of sepals and leaves first. The opening of leaf buds follows a few days later. The leaf parts are most susceptible when young, becoming quite resistant to infection as they approach maturity, though sparse development of the fungus may occur on the dorsal surface of old leaves. Warm, relatively dry weather hastens host development and is relatively unfavorable to the development and dissemination of primary inoculum. Cool, moist weather has an opposite effect. The development of primary infection is influenced greatly, therefore, by the environment during early spring. A thorough study of epidemiology of the disease in Wisconsin has been reported by Keitt and Jones (11).

Sporulation on primary lesions occurs readily over a wide range of conditions without presence of rain. There is, therefore, usually an abundant source of secondary inoculum once primary infection has occurred. Conidia ordinarily begin to appear before the lesion is macroscopically visible. In contrast to the ascospores, conidia depend upon water for dispersal and dissemination.

The most critical time for the development of epidemics is from the time of opening of fruit buds until petal fall. In northern areas where cool, moist springs are common, the environal conditions are likely to be most favorable at this time. Early infection of sepals provides a very favorable source of secondary inoculum for infection of the young fruits. The autumn period is next most important; late infection of fruit occurs then if a supply of leaf lesions has been permitted to develop. Although fruits of some varieties increase in resistance with age, infection in many other varieties may occur well up to harvest. Incipient, invisible infections at this time may result in development of lesions in storage (3).

Ascospores and conidia germinate in contact with the host surface and usually form an appressorium (Fig. 95). A fine penetration tube emanates from the latter, occasionally from a spore or an undifferentiated germ tube, by way of a small pore in the wall which is in contact with the host surface (19). The infection hypha penetrates the cuticle directly without any demonstrable solvent effect on the latter. A hypha of normal diameter grows from the end of the penetration tube and develops beneath the cuticle in close contact with the outer cellulose wall. The fungus develops into a microscopic stroma in the subcuticular position with little or no deleterious effect upon the epidermal and palisade cells of the leaf. About the time the lesion becomes macroscopic, the underlying cells show signs of impoverishment, the contents gradually become aggregated, and

eventually the cells collapse and die. The fungus remains largely in the subcuticular position, deriving its nourishment from palisade and eventually from mesophyll cells. After leaf fall, growth ensues through the intercellular spaces from that portion of the stroma in which the cells contain considerable amounts of protoplasm.

Varietal Resistance. Apple varieties differ markedly in their reactions to given isolates of the pathogen. In all cases the fungus penetrates the cuticle. In some cases of high resistance the epidermal cells collapse to form a necrotic spot. Gradations between this and the reaction described in the previous section occur in various isolate-variety combinations. No varieties of commercial value are sufficiently resistant to per-

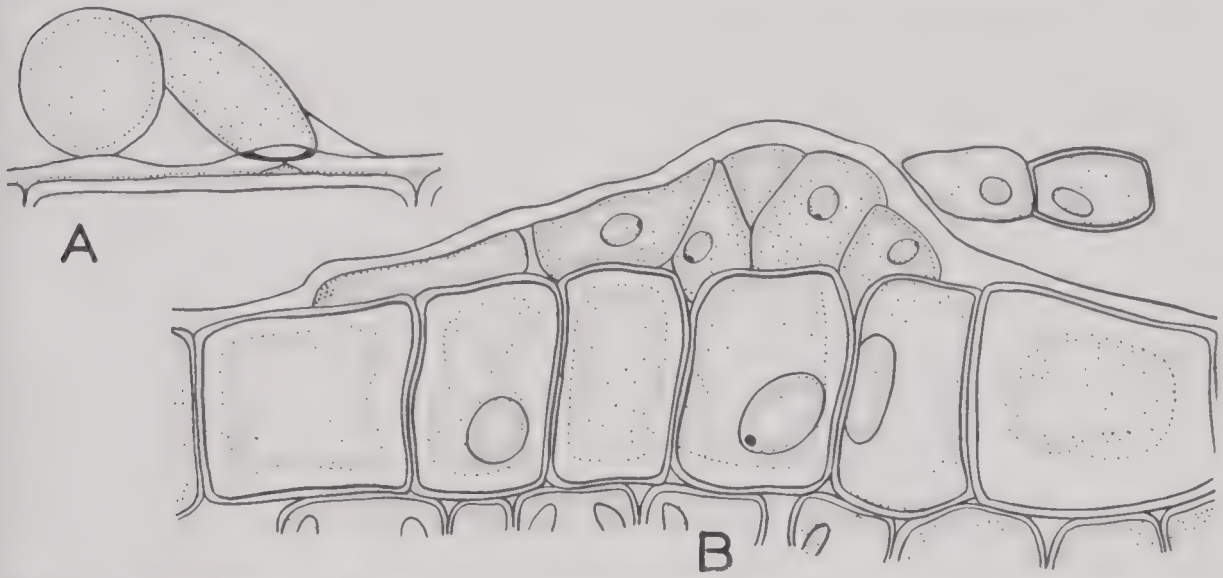

FIG. 95. Apple scab. *A*, penetration of cuticle of leaf; *B*, subcuticular mycelium in infected leaf. (*After Nusbaum and Keitt.*)

mit the omission of a protective fungicidal program in regions where the disease develops in epidemic form. Breeding of apples for scab resistance has been carried on in Germany (23). Evaluation of various wild and cultivated stocks has been made, and breeding programs have been initiated, by Hough and Shay (9, 10, 24) in the United States and by Hockey and Eidt (8) in Canada.

Control. Control of apple scab with fungicides began soon after the discovery of Bordeaux mixture by Millardet. This fungicide was very successful except for injury such as russeting of the fruit. Lime-sulfur, revived early in this century, was used for scab control with success, but limitations were also found in the form of fruit and foliage injury during hot midsummer weather. Sulfur dust was next studied but without general success. Wettable sulfurs and Fermate are now used widely for the midseason sprays. See Chap. 16 for discussion of the fungicides.

Keitt and associates have studied the possibilities of reducing the

primary inoculum by eradication of the fungus in infected leaves toward the close of the growing season and on the orchard floor before bud break in the spring (4, 12, 14, 21, 22). Elgetol or Krenite is recommended for the ground spray, which greatly reduces the potential inoculum and improves the results of the later protective-fungicide program where satisfactory control on particularly susceptible varieties is not always obtained.

The spray schedule for any given locality should follow the recommendations of the nearest experiment station (5–7, 11, 14, 18, 21). As an example, the recommendations for the peninsular and lake-shore areas of Wisconsin are as follows:

1. Ground spray of 600 gal. per acre of Elgetol or Krenite ($1\frac{1}{2}$ per cent) after surface water is off and before bud break.
2. Prebloom sprays of lime-sulfur, 1–60, starting at the dormant stage and continuing at about weekly intervals through bloom.
3. Five or more postbloom sprays of milder fungicides such as wettable sulfur or Fermate, beginning when most of the petals are off and continuing at 10-day intervals.
4. Final spray of lime-sulfur.
5. Incorporate suitable insecticide with the protective sprays.

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BITTER ROT OF APPLE

Bitter rot was described in Europe in an unpublished manuscript dated in 1829. Berkeley described the disease and the pathogen in England in 1856. The disease occurs on apple throughout the world. It is restricted as a major disease, however, to relatively warm, humid areas. In the United States it causes severe losses only in the southern zone of commercial apple production, roughly south of the fortieth parallel and east of the Continental Divide. The pathogen affects a wide range of hosts, particularly as an incitant of decay of ripened fruits.

Symptoms. When the environment is favorable, the fruits of particularly susceptible varieties may be affected when young and green. In other varieties fruits seldom become diseased until after midseason. When weather is warm and moist as the crop approaches maturity and when

sources of infection are numerous, it is not uncommon for an entire crop to be almost completely lost on the trees within a very few days. The first sign of the disease on apple fruits is a faint light brown discoloration beneath the skin growing in surface diameter and in a cone-shaped area toward the core. As the roughly circular surface lesion reaches about 3 mm. in diameter, it begins to become increasingly depressed (Fig. 96). As it reaches about 1 cm. in diameter, tiny, raised, black dots appear beneath the cuticle, giving rise to fruiting bodies (acervuli), which rupture the cuticle and in moist weather produce sticky pink masses of spores, which may protrude in tendril-like cirri. The spore masses may be arranged in well-defined rings. If they are washed off by rain, the underlying stromata give a dark, almost black, color to the spot. As the lesions increase in size, the entire fruit may be rotted, the decayed tissue being dry and pulpy unless secondary soft-rot organisms enter. Rotted fruits drop at all stages, some clinging as mummies throughout the winter.

FIG. 96. Bitter rot of apple. (*Courtesy of U.S. Department of Agriculture.*)

Cankers occur on limbs 2 or more years of age. They are oval in outline, and the wood beneath is dead and dry with dead bark and cambium adhering. Fissures develop in the dead bark, and the surface of the canker is depressed. Cankers which start on young, vigorously growing branches are usually corked out promptly and do not survive until the next season. Those on older limbs, particularly those on varieties especially susceptible to limb infection, may survive for many years, although the fungus is usually dead after about the third year. The wood and rays are brown in color. Gum is abundant in bark and cambium, and it occurs to some extent in wood and rays. A callus layer forms around the outer border of the canker.

The Causal Organism. *Glomerella cingulata* (Ston.) Spauld. & Schrenk, 1903.

SYNONYMY:

Conidial stage:

Septoria rufo-maculans Berk., 1854.

Gloeosporium fructigenum Berk., 1856.
Gloeosporium laeticolor Berk., 1859.
Ascochyta rufo-maculans Berk., 1860.
Gloeosporium versicolor Berk. & Curt., 1874.
Gloeosporium rufo-maculans (Berk.) Thuem., 1879.
Gloeosporium cingulatum Atk., 1892.

Ascigerous stage:

Gnomoniopsis cingulata Ston., 1898.
Gnomoniopsis fructigena Clinton, 1902.
Glomerella rufomaculans Spauld. & Schrenk, 1903.

The conidial stage was described under various names as shown above. Shear and Wood (13) considered the species an omnivorous one and brought together under one species name many forms from a long list of hosts. The first ascigerous stage found on apple was that reported from Illinois by Clinton in 1902. This was first placed in *Gnomoniopsis*. Schrenk and Spaulding pointed out that this generic name had been used previously for another group of species, and they created the generic name *Glomerella*, designating the apple fungus as *Glomerella rufomaculans*. Since Shear and Wood (13) regarded the fungus on apple to be synonymous with similar forms on other hosts, the proper binomial becomes that of the first ascigerous form of the species to be recorded. This was the form described in the imperfect stage on privet by Atkinson in 1892 as *Gloeosporium cingulatum* and of which the ascigerous stage was described by Stoneman as *Gnomoniopsis cingulata* in 1898.

The mycelium is hyaline when young, branched, producing dark-walled chlamydospores and dark stromata, on which acervuli are formed beneath the epidermis, rupturing the latter (Fig. 97). Setae are irregular in appearance according to Shear and Wood (13). Conidia are subhyaline; pinkish en masse; variable in shape, mostly oblong or cylindrical, some slightly curved; 3.5 to 7 by 10 to 28 μ, mostly 4 to 5 by 12 to 16 μ. Dark-walled appressoria are produced at the ends of germ tubes. Stromatoid cushions support immersed and more or less compounded subspherical perithecia. Asci are subclavate, often slightly pedicellate, fugacious, 55 to 70 μ in length. Ascospores are allantoid, hyaline, continuous, 3.5 to 5 by 12 to 22 μ; they are difficult to distinguish from the conidia.

The cytological details of ascus formation are reported by Lucas (7). The young ascus contains two haploid nuclei which fuse to form the diploid stage. Meiosis occurs in the first two divisions of the fusion nucleus. The haploid nucleus appears to have four chromosomes.

Edgerton was the first to point out that when certain strains were mated in petri-dish cultures, perithecia formed promptly at the line of contact. Further studies in his laboratory (4, 5, 7, 8, 14, 15) have shown that from

the wild type so-called "plus" and "minus" strains may be isolated which produce perithecia in abundance when mated. That the fungus is not strictly heterothallic is shown by the fact that some "plus" and some

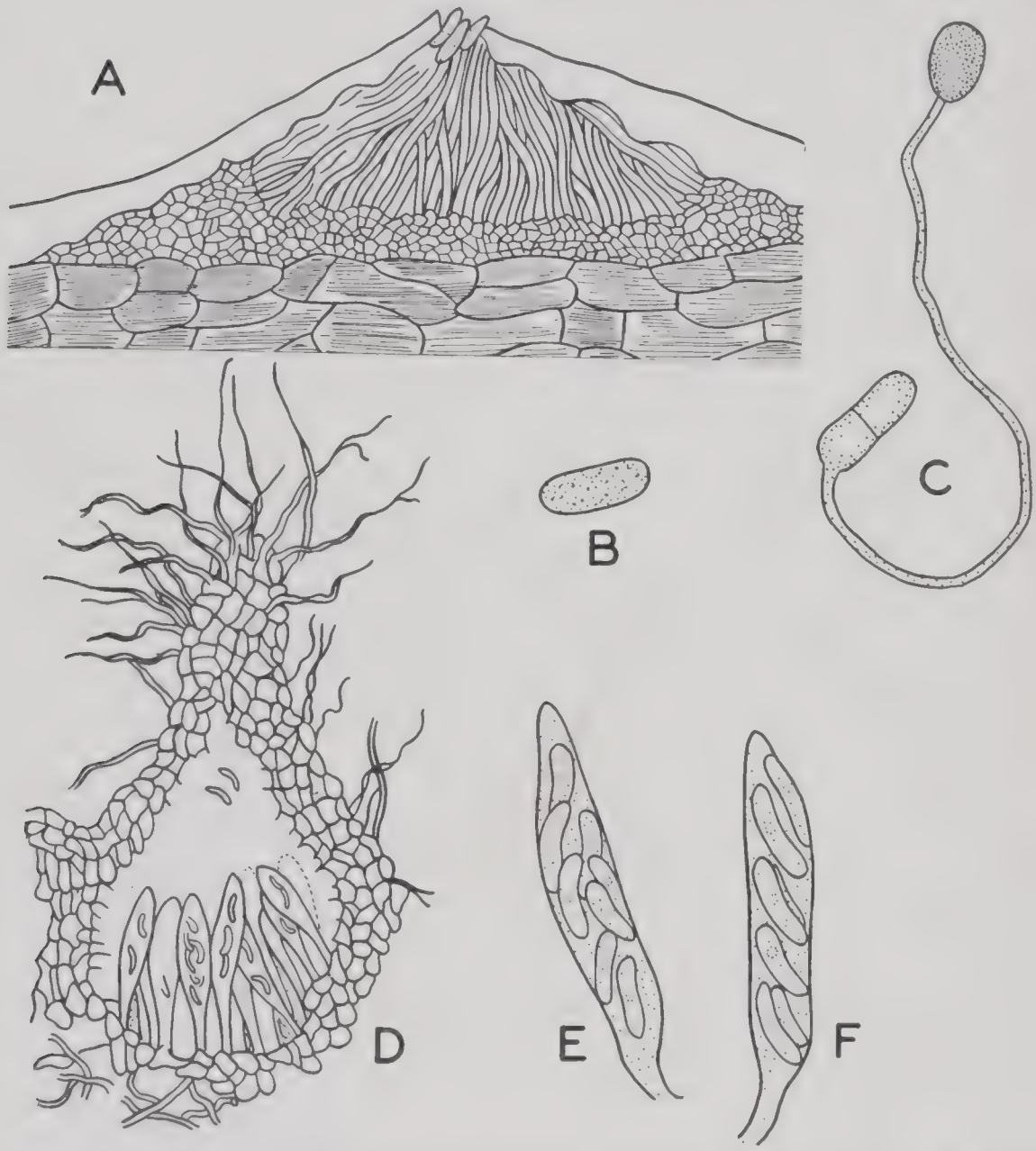

FIG. 97. *Glomerella cingulata*. *A*, acervulus on apple fruit; *B*, conidium; *C*, conidium, having become septate during germination; appressorium at tip of germ tube; *D*, perithecium; *E*, *F*, asci. (*A–C*, *E*, *F*, *after Clinton; D after Stoneman.*)

"minus" strains produce functional perithecia without mating. Conidial lines which produce no perithecia without mating were also secured. By conducting genetic analyses from perithecia formed at the margin of two cultures differing in a given character, it was shown that fertilization between the two strains occurred. When a non-perithecium-forming conidial strain was mated with a "plus" type, slow development of perithecia

occurred while more rapid development occurred when it was mated with a "minus" type. In contrast with *Venturia inaequalis* (p. 329) then, some strains of this fungus are homothallic; others are heterothallic. However, mating of some homothallic lines results in cross-fertilization and a greater production of perithecia. So far, two pairs of alleles, one at each of two loci, have been shown to control production of fertile perithecia. Partial self-incompatibility in some of the minus strains is conditioned by a gene epistatic to the self-incompatibility gene at one of the loci. Self-incompatible lines are shown to arise as mutants from a self-compatible line. Other genetic studies in *Glomerella* are reported by Andes (1) and Hüttig (6).

Disease Cycle. The sources of primary inoculum are perennial cankers, deadwood, and mummied fruits of the previous year which remain on the tree. While the ascigerous stage may form in mummied fruits and in deadwood, it is not essential or important since the fungus hibernates successfully as stromatoid mycelium and produces conidia in abundance when moisture and temperature become favorable. The desiccated conidial masses are capable of surviving for several months. Edgerton described two strains, one fast-growing with an optimum of 29 to 30°C., the other slow-growing with an optimum of 25°. Brooks and Cooley (2) reported an optimum of 25°. The conidia are dependent on meteoric water for dissemination, and both insects (flies) and birds are known to disperse the spores. Where spores are borne in cirri, they may be dislodged and disseminated by wind. Infection occurs directly through the cuticle after an appressorium is formed, as described for *Colletotrichum* (p. 274). Under optimum conditions the fungus completes the conidial cycle within a very few days, and this facilitates very rapid development of epidemics.

Control. Removal of deadwood and mummies is the first essential measure. When this has been done, protective sprays usually give good control. The copper fungicides (p. 331) tend to be more effective than sulfur compounds for the control of this disease.

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ENDOTHIA CANKER, OR BLIGHT OF CHESTNUT

Chestnut blight is a disease which is unique in that within three decades after its discovery it had, for all practical purposes, destroyed the valuable stands of American chestnut in the Appalachian range of the eastern United States. It was found first in the zoological park of New York City in 1904. By 1914 it was recorded as far south as North Carolina and nearly as far west as the western border of Pennsylvania, while damage was severe throughout eastern Pennsylvania, Maryland, and northern Virginia. Gradually the heaviest stands of chestnut extending from Maryland to Georgia were destroyed. Many species of *Castanea* are potential hosts, but in general Asiatic species are rather highly resistant.

The disease was discovered in China in 1913 and later in Japan. There is good reason to believe that the pathogen was introduced into the United States from the Orient with nursery stock. It has been found on ornamental and nut species of chestnut in the Middle West and in the Pacific coast states, as well as in Ontario and British Columbia. It was observed in Italy for the first time in 1938.

Symptoms. The first conspicuous evidence of chestnut blight is seen in midsummer as brown shriveled leaves on twigs that have been girdled by bark lesions. Such foliage, and burs if present, may cling throughout the winter. Water sprouts occur commonly below dead branches. The cankers which induce the killing occur on twigs, branches, and the main trunk (Fig. 98). They are ellipsoidal with fairly regular

margins. On relatively new canker tissue pycnidia appear and in moist weather exude spores in long yellow cirri. In older canker tissue reddish-brown pustule-like areas up to 2 mm. in diameter appear by rupturing of the outer bark. These are the ascigerous stromata. On rapidly growing branches the tissue at the margin of the canker may consist of hypertrophied cells, giving a raised border. As the cankers become older, the bark splits and falls away. If the bark is peeled back, the fan-shaped mycelial mats of the fungus on the cambium are characteristic signs of the disease.

FIG. 98. Chestnut blight lesion on branch, showing cirri of pycnospores. (*Courtesy of U.S. Department of Agriculture.*)

The Causal Organism. *Endothia parasitica* (Murr.) P. J. Anderson & H. W. Anderson, 1912.

SYNONYMY:

Diaporthe parasitica Murr., 1906.

Valsonectria parasitica (Murr.) Rehm, 1907.

Endothia gyrosa var. *parasitica* (Murr.) Clinton, 1912.

The mycelium is multinucleate, monopodially branched, irregular in diameter, taking on a yellowish color en masse. On the host the mycelial mats in bark and cambium are extensive and conspicuous.

Pycnidia are globose with walls of rather loosely tangled hyphae forming beneath the surface of the smooth back of young cankers (Fig. 99). The conidiophores (20 to 40 by 1.5 μ) cut off successively spores which under favorable conditions are forced out of the ostiole when the bark is ruptured. Older pycnidia are surrounded by a mature stroma, one fruiting body to a stroma, and the cavity of the pycnidium is irregular and convoluted. Pycnospores (average 1.3 by 3.6 μ) are continuous and oblong with rounded ends.

On cankers which are a year or more old there appear orange-colored or reddish-brown erumpent stromata, the outer cells of which are packed into a rind-like layer. Numerous perithecia form within the stroma, and each has a long neck which extends to the surface where the papillate ostiole is visible as a black speck (Fig. 100). Asci average about 9 by 51 μ

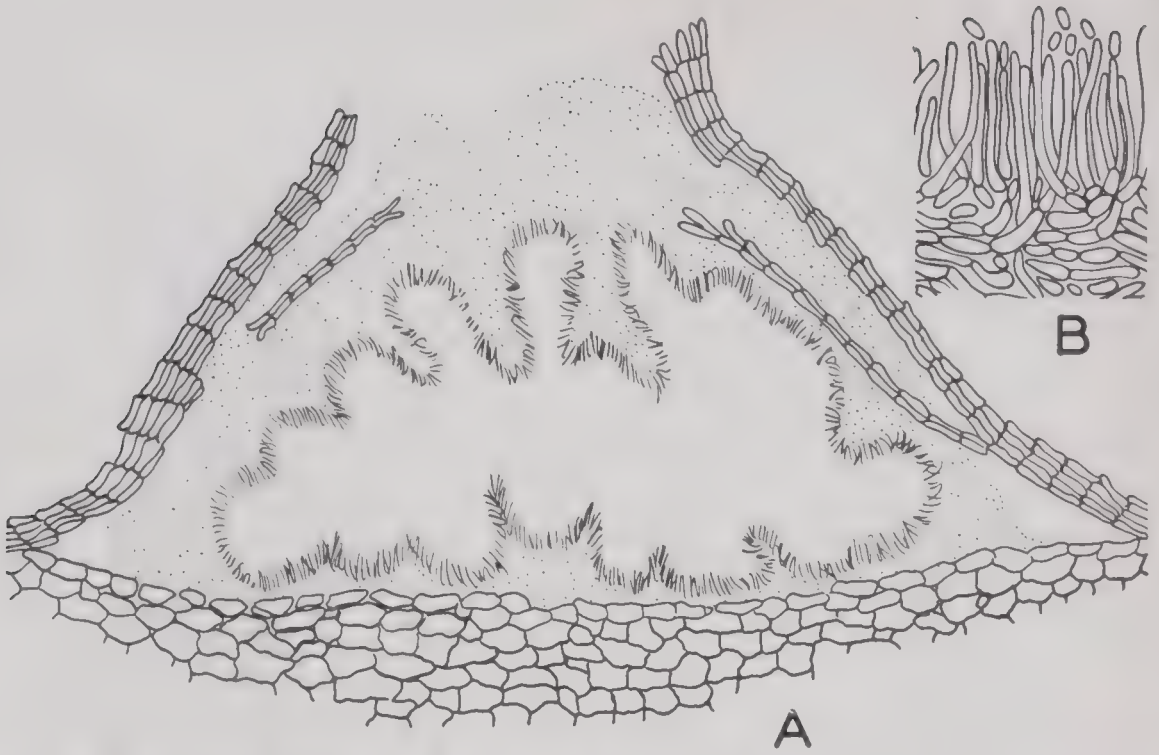

FIG. 99. *Endothia parasitica*. *A*, cross section of pycnidium; *B*, conidiophores bearing pycnospores. (*A after Heald; B after Anderson and Rankin.*)

FIG. 100. *Endothia parasitica*. *A*, cross section of stroma showing perithecia; *B*, ascus. (*After Heald.*)

and contain eight ascospores. The latter are oblong with rounded ends, two-celled, constricted at the septum, each cell having two to four nuclei; the spores average about 4.5 by 8.6 μ in size.

Disease Cycle. The fungus subsists in cankers and as a saprophyte on dead plant parts. Pycnospores and ascospores are produced throughout spring and summer. Pycnospores do not germinate in pure water, but when favorable nutrient is present, they first swell to four or five times the original length and diameter and then produce two germ tubes in about 12 hr. at the optimum temperature (about 30°C.). Ascospores swell somewhat before germination and produce germ tubes more promptly than the pycnospores. The optimum temperature for germination of ascospores is about 20°. The pycnospores overwinter in the cirri and remain viable in the soil for about 4 months. The ascospores may remain viable within perithecia for a year or more, if conditions for ejection do not occur. Penetration takes place through wounds in the bark, and infection follows only during summer months; leaves are not infected.

The rapid spread of the pathogen is circumstantial evidence that dissemination is carried out effectively. The pycnospores are disseminated by spattering water, wind-blown rain, insects, and birds. Ascospores are discharged into the atmosphere and carried by air currents. They have been caught 380 ft. from the point of ejection and probably are carried much farther by wind. Nursery stock and infected logs are a ready means of wide distribution of the pathogen.

Trees of all ages and sizes are eventually killed. In general, the roots survive and continue to produce new sprouts, which, in turn, become infected.

Control. Now that the pathogen has become established and has successfully prevented the regeneration of the forests of American chestnut, the only means of control rests with the possible development of a suitable resistant form. For nut production satisfactory control may be had by using resistant Asiatic varieties. These highly resistant forms, however, are not suitable to replace the native species of the Appalachian forests. A breeding program with the objective of producing resistant lines of a suitable forest-tree type has been under way since the early period of chestnut blight in the United States. As yet no resistant individuals within the American chestnut species have been found. The most promising resistant species are the Japanese chestnut (*Castanea crenata* Sieb. & Zucc.) and the Chinese species (*C. mollissima* Blume). Neither of these has the type of tall growth needed in a forest tree. In the F_1 hybrid of American and Japanese chestnuts the growth habit and resistance are intermediate, indicating that both characters are polygenic.

It is to be expected that a long period of years will be required before suitable resistant forest trees are secured by this means.

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GIBBERELLA DISEASES OF CORN AND SMALL GRAINS

Two species of *Gibberella* (*G. zeae* and *G. fujikuroi*) are pathogens of corn, and the former is important on small grains. Head blight (scab) and seedling blight and foot rot occur on barley, wheat, rye, and some varieties of oats. Seedling blight, root rot, stalk rot, and ear rot occur on corn. Head blight was described as early as 1884 in England and in the United States in 1891. The connection between seedling blight and head blight of wheat was shown by Selby and Manns (7) in 1909. Arthur (7) suggested a possible relation of seedling blight of corn and wheat scab in 1891, but it was not clearly demonstrated until the report of Hoffer *et al.* (18) in 1918. These diseases have a world-wide distribution. They are most important in humid regions. The disease incited by *G. zeae* on corn is most common in the eastern and central portions of the United States, while those incited by *G. fujikuroi* and its variety *subglutinans* are common also in southern portions of this country. The latter fungus appears as a pathogen in boll rot of cotton, in stalk rot of sorghum, in pokkah-bong of sugar cane, as well as on banana, pineapple, and Manila hemp. Stalk rot and ear rot of corn incited by *Gibberella* may at times be difficult to distinguish from that associated with infection by *Diplodia zeae* (p. 290).

One of the unique characteristics of the disease incited by *G. zeae* is that the causal fungus growing in the developing kernels produces a by-product which acts as an emetic when the grain or products manufactured from it are used as feed for swine or as food by humans. This first came to attention in eastern Siberia (15), where bread made from affected wheat has long been referred to as "intoxicating bread," since its use results in dizziness, nausea, and vomiting. It has been important from time to time where affected grain fed to hogs brings on emesis. After a severe

epidemic of barley scab in the Middle West in 1928 much sickness in swine resulted in the United States and in Germany where grain from the affected area was used (4, 21).

Symptoms. Head blight, or scab of small grains, appears during the period between flowering and maturity. One or more of the spikelets turn straw-colored. In moist, warm weather pink to red fluffy growth consisting of mycelium and conidia appears, sometimes overrunning the entire spike. Purplish perithecia may develop promptly on the floral bracts in warm, moist weather. Kernels become wrinkled, and discoloration varies with the environment and with the stage of development of the kernel when it is infected. Wheat kernels become white, pink, or light brown and have a rough, flaky surface. Rye kernels are dark brown to carmine red depending on the degree of disease development. Barley and oats are similarly affected, but they are less conspicuous because of the hulls, which are darkened somewhat in the case of barley but little or not at all with oats.

In small grain and corn seedlings the developing embryo may be invaded, turn reddish brown in color and die before either roots or plumules elongate. In other cases the plant may be attacked after emergence, the pathogen usually growing from the invaded endosperm into the seedling internodes and the roots. Plants attacked later may first become dwarfed and chlorotic and later blight. In corn the lesions usually occur on tap and lateral roots and in the scutellar node and in the internode between the scutellum and the coleoptile (mesocotyl). The lesions are light brown and water-soaked, turning darker in color with age. When *G. fujikuroi* is concerned, the weakened plants have less prominent root lesions and less frequent invasion of the scutellum node and the mesocotyl. Seedling blights and root rots are incited by a large number of fungi, of which the species of *Gibberella* are only two. Root rot in small grains is more commonly incited by *Fusarium culmorum* (W. G. Smith) Sacc. *Pythium* seedling blight and root rot has been discussed in Chap. 6, and *Diplodia* blight and root rot is described in Chap. 7.

Root rot and stalk rot of corn develop during the growing season. The progress of this phase of the disease is not greatly different from that described for *Diplodia* root rot and stalk rot (p. 290). The lesions tend to be reddish in color; premature ripening occurs; and stalk breaking is common.

Ear rot of corn starts usually at the tip rather than at the butt as in *Diplodia* ear rot (p. 290). Husks are affected also. With *G. zeae* a pink to reddish rot progresses down the ear; with *G. fujikori* scattered individual kernels are affected, or the entire ear may be overrun. Kernels show various degrees of rotting. They may be pink to red in color, owing to

conspicuous extramatrical mycelium, or show merely a shrunken lusterless pericarp. Many infected ears and seeds show no signs, and the fungi can be detected only upon germination in blotters or by plating on agar.

The Causal Organisms.

1. *Gibberella zeae* (Schwabe) Petch.

SYNONYMY: *Gibberella saubinetii* (Mont.) Sacc.[1]
Conidial stage: *Fusarium graminearum* Schwabe.

2. *Gibberella fujikuroi* (Saw.) Wr.

SYNONYMY: *Gibberella moniliformis* (Sheldon) Wineland.[1]
Conidial stage: *Fusarium moniliforme* Sheldon.

3. *G. fujikuroi* var. *subglutinans* Edwards.[1]

Conidial stage: *Fusarium moniliforme* var. *subglutinans* Wr. & Reinking.

The mycelium of *G. zeae* is septate, branching, and white to pink en masse. The macroconidia are typical of *Fusarium* (see p. 255), sickle-shaped, hyaline, up to five-septate, 4.3 to 5.5 by 41 to 60 μ (Fig. 101). They are produced very promptly under favorable conditions on sporodochium-like clusters of short conidiophores, where they form acrogenously. Septation often is completed after the conidium is pushed off to make room for a succeeding spore. After an initial crop of conidia is formed, the latter may germinate to form a stroma from which, after a few weeks, typical sporodochia arise (9). Microconidia and chlamydospores do not occur. The perithecia are ovoid to subconical, purplish black to dark blue, scattered over the host surface, somewhat embedded, smooth at the base with protuberant projections near the apex. Asci are cylindrical, tapering toward the base, slightly curved, hyaline. Ascospores are usually eight per ascus, fusiform, slightly curved, hyaline, mostly three-celled, 3.4 to 5.0 by 20 to 30 μ.

Macroconidia in *G. fujikuroi* are three- to five-septate, 2.9 to 3.5 by 30 to 56 μ, but rare. Microconidia are numerous; borne basipetally on simple to verticillate conidiophores, adhering to form chains or slipping aside to form clusters; obovoid to ellipsoid, 2 to 3.5 by 5 to 10 μ. Perithecia are scattered to gregarious, ovoid to subconical, free or embedded; ostiolate; 225 to 300 by 300 to 375 μ; asci are straight; ascospores fusiform to ellipsoid, rounded at ends and often constricted at septa; pale ochraceous salmon en masse; mostly one-septate; 3.9 to 4 by 15 to 19 μ (38). *G. fujikuroi* var. *subglutinans* differs from *G. fujikuroi* in that it produces

[1] A more complete synonymy is given by Dickson (8).

microconidia in clusters but not in chains; and the ascospores are more blunt and of a more short elliptical shape.

Considerable variability in cultural characters and in virulence has been observed in these as in many species of *Fusarium* (14, 16, 25, 34).

Disease Cycle. The fungi overwinter in seed or on infected debris, particularly in infected cornstalks. Perithecia may appear in the early fall, but in northern states they mature most commonly in the spring. Air-borne ascospores thus serve as the chief primary inoculum. Macro- and microconidia are formed abundantly on infected plant parts in moist warm weather and serve as the air-borne secondary inoculum.

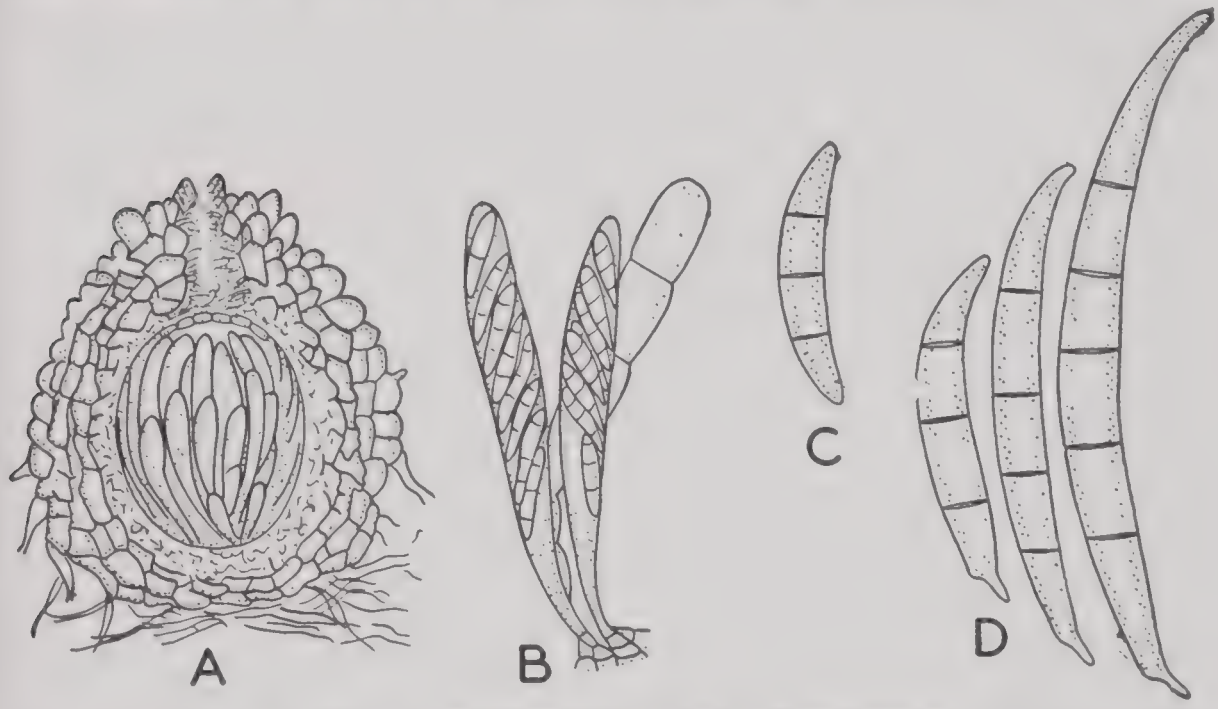

FIG. 101. *Gibberella zeae*. *A*, perithecium; *B*, asci; *C*, ascospore; *D*, macroconidia. (*After Wollenweber.*)

Infection of heads of small grains occurs chiefly through the floral parts, moist warm weather being essential for epidemic development. Pugh *et al.* (28) showed that when flowers were invaded at flowering time, the kernels which develop later are invaded throughout. As the wheat kernel grows, however, the semipermeable membrane on the inner integument becomes increasingly resistant to penetration. Andersen (1) showed that susceptibility in wheat increased from the flowering to the postflowering stages and decreased at late stages of seed development.

The details of penetration and host-parasite relations in the corn seedling have been worked out by Pearson (27) for *G. zeae*. As the adventitious roots arise in the pericycle of the stele and traverse the cortex, they form channels through which the fungus invades. The latter proceeds through the intercellular spaces of the cortex, invading the cells in the

older portion of the lesion. The intercellular mycelium causes swelling of the cell walls in some regions. When a hypha penetrates from cell to cell, it often becomes greatly constricted at the point of penetration; a fine filament passes through the wall, and the mycelium then regains the original diameter as it enters the next cell cavity. In the coleorhiza, plug-like structures occur at the surface between the epidermal cells. They appear not to be cuticularized as is the rest of the epidermal layer. The pathogen penetrates the coleorhiza through the plugs, which are apparently readily dissolved by fungus secretions. The fungus develops in the epidermal wall, which swells considerably, and it also proceeds intercellularly. Cellular penetration follows after the coleorhiza has been rather completely invaded, at which time the fungus may proceed into the primary root. The relation of *G. fujikuroi* to young corn seedling tissue is described by Voorhees (37).

Soil temperature is important in determining the extent of seedling blight of wheat and corn incited by *G. zeae*. Dickson (7) showed that the fungus grew on agar at a range of 3 to 32°C. with an optimum at 24 to 28°. Wheat grows best at a relatively low soil temperature while corn develops best at relatively high soil temperatures. Seedling blight of wheat develops little or not at all at 12° if other factors for growth are favorable, and severity of the disease increases with temperature as high as 28°. With corn, on the other hand, the most favorable range for seedling blight is 8 to 20°, while none occurs above 24° (Fig. 172). It is obvious that the effect of temperature on disease development is dependent upon the reaction of the host rather than of the pathogen. When the temperature is most favorable for the host, it withstands the pathogen most effectively. Eckerson and Dickson (12) attributed the resistance or susceptibility of the plants, as influenced by temperature, to the chemical composition of cell walls and the nature of food available to the pathogen. Wheat seedlings grown at low soil temperature are high in available carbohydrates and low in available nitrogen, while the cell walls even in the early seedling stage are cellulose soon impregnated with lignin. At high soil temperatures wheat seedlings have little or no available carbohydrate and are high in available nitrogen, while the cell walls are composed of pectic materials, cellulose being absent until after photosynthesis begins. The cell walls are, therefore, the most resistant and the food reserves least attractive to the fungus in the low-temperature plants. Corn seedlings show the reverse situation at high and low temperatures with reference to cell walls and reserves, and high-temperature plants are correspondingly the most resistant.

Varietal Resistance. Although differences in susceptibility of barley and wheat varieties to head blight are known to exist (6, 22, 29), no com-

mercially satisfactory, highly resistant varieties have been developed. Inbred lines of corn are known which are highly resistant to seedling blight (10). Hoppe (19) and McIndoe (26) have studied the inheritance of such resistance, and their results indicate that it is polygenic. Senn (30) compared sweet and starchy kernels from the same segregating ears and found that the sugary kernels were practically always significantly more susceptible to seedling blight. There are difficulties, however, in testing breeding progenies for inherent resistance to this disease in the seedling stage, since the conditions under which the seed is matured have a very marked influence upon the resistance of the resulting seedlings to seedling blight (11, 19). This may outweigh and obscure the expression of inherent resistance, as shown by Hayes *et al.* (17). Corn breeders have succeeded, however, in producing many hybrids which are relatively high resistant to *Gibberella* and *Diplodia* stalk rots (23).

Control. Since the fungi overwinter on debris, sanitary measures are helpful in reducing spring inoculum. Where small grain follows corn, complete turning under of stalks and stubble is desirable in areas where scab is inclined to be prevalent. Seedling infection results most commonly from seed-borne inoculum. Seed protectants are important. This phase is discussed under *Diplodia* disease of corn (p. 290) and in Chap. 16. In any area where *Gibberella* stalk rot is prevalent, breeders usually evaluate hybrids as to stalk-rot resistance. The best control is to choose from such lines that are available and otherwise adapted to the area concerned.

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ERGOT OF GRAINS AND GRASSES

Ergot is a disease which affects only the flowering parts of many wild and economic species of the grass family. Among economic cereals it is most important on rye. There are numerous species within the genus *Claviceps* which may be concerned as pathogens, but the most common one is *Claviceps purpurea* (Fr.) Tul. The disease is world-wide in occurrence.

The economic importance of the disease has two aspects. Affected flowers produce ergot sclerotia instead of kernels and thereby yield is reduced. Ordinarily, this reduction is not great, although losses up to 20 per cent have been reported. The other phase concerns the fact that sclerotia of *C. purpurea* contain alkaloidal materials which are injurious to livestock if they are fed grain containing the fungus bodies. The latter may also cause an alkaloidal poisoning of humans if they remain as a contaminant when grain is converted to bread flour. A low tolerance of ergot is the rule, therefore, when grain is graded for food, feed, or seed purposes. The ergot sclerotia may be removed, but this adds to the cost and is one of the bases of scoring off grain which contains them.

The alkaloids extracted from sclerotia are a recognized drug, and the commercial source of this material is the sclerotia removed from grain at the elevator. The drug came into use long before the nature of the disease was understood.

Because of the conspicuous nature of the disease, it was observed and recorded by the Greeks and Romans, making it one of the oldest known plant diseases. Many interpretations were placed upon the nature of the sclerotium in the period previous to 1850. The function of the sclerotia in production of the ascigerous stage was first noted by Tulasne in 1852. The full life history of the fungus was established by Kühn in 1863.

A large number of cereals and grasses are host plants.

Symptoms. The conspicuous phase of the disease is the formation of sclerotia in the position of kernels in the spike. They are two or three times the length of the kernel, curved, and grayish violet in color (Fig. 102). The surface has a dusty appearance and may be corrugated longitudinally.

Traverse or longitudinal cracks with reddish margins sometimes occur, exposing a light-colored interior. A noticeable odor is given off.

The ergot stage is preceded by a honeydew conidial stage which consists of a sticky mass on the young ovary. This comprises the primary infection, and in affected ovaries growth of the sclerotium begins, eventually preempting the position of the ovary within the glumes and carrying the conidial stroma and sometimes the stamens upward on its tip as it enlarges.

FIG. 102. Ergot of rye. *Left*, conidial stage; *right*, sclerotial stage. (*After Dickson.*)

The Causal Organism. *Claviceps purpurea* (Fr.) Tul., 1853.

SYNONYMY:

Sclerotial stage:

Clavaria solida Munchhauses, 1765.
Clavaria clavus Schrank, 1789.
Clavaria secalina Paulet, 1793.
Sclerotium clavus DC., 1815.

Conidial stage:
Spermoedia clavus Fr., 1822.
Sphacelia segetum Lév., 1826.
Ergotetia arbortifaciens Quackett, 1841.
Oidium arbortifaciens (Quackett) Berk. & Broome, 1851.
Ascigerous stage:
Sphaeropus fungorum Paulet, 1793.
Sphaeria purpurea Fr., 1822.
Sphaeria entomorhiza Schumacher, 1823.
Sphaeria capitata Schumacher, 1823.
Kentrosporium purpurea (Fr.) Wallr., 1844.
Cordyceps purpurea Fr., 1846.
Cordiliceps purpurea (Fr.) Tul., 1851.

The sclerotium in the soil gives rise to stromata, which form as globose, compact white bodies about $^1_2$ mm. in diameter. They are extended to the surface of the soil by growth of a stipe, which passes through color shades of pale yellow, red, and finally purple or violet. Up to 30 stromata may form from a single sclerotium. The heads (**sphaeridia**) are yellowish at first, becoming flesh-colored to pale fawn as they mature. The ascogonia and antheridia are formed in the peripheral layer of the sphaeridium, and after fusion, ascus formation proceeds in the usual manner (Fig. 103). The perithecia, embedded in the sphaeridium, have ostioles which project to the surface, giving the latter a slightly warty appearance. The flask-shaped perithecia contain long, curved, hyaline asci, narrowed at both ends and surrounded by paraphyses which are thickest at the apical end. The ascus contains eight long (50 to 76 μ), filiform, slightly curved, hyaline, continuous or septate ascospores.

In the conidial stage numerous short unbranched conidiophores are produced in close array over the intricately folded and convoluted surface of the so-called sphacelial stroma, which is formed in or on the ovary remains prior to development of the sclerotium. Small, unicellular, hyaline conidia (3.5 by 7 μ) are abstricted in succession from the tips of the conidiophores. These spores are embedded in a copiously produced sticky fluid known as "honeydew."

Disease Cycle. The fungus overwinters as sclerotia, which, in the spring, give rise to stromata and ascospores, the latter becoming the primary inoculum. Krebs (7) found that after the sclerotia had been exposed to a freezing temperature for 1 month, stromata were produced most rapidly at 9 to 15°C. and were inhibited at 18° and above. Perithecial formation was favored by somewhat higher temperatures. The host is susceptible in the flower tissues and only for a short period following anthesis. The coordination of ascopsore development and the flowering period of the

host is, therefore, essential to epidemic development. Lack of such coordination often precludes or reduces infection. Those species and varieties in which glumes tend to remain closed during flowering largely escape

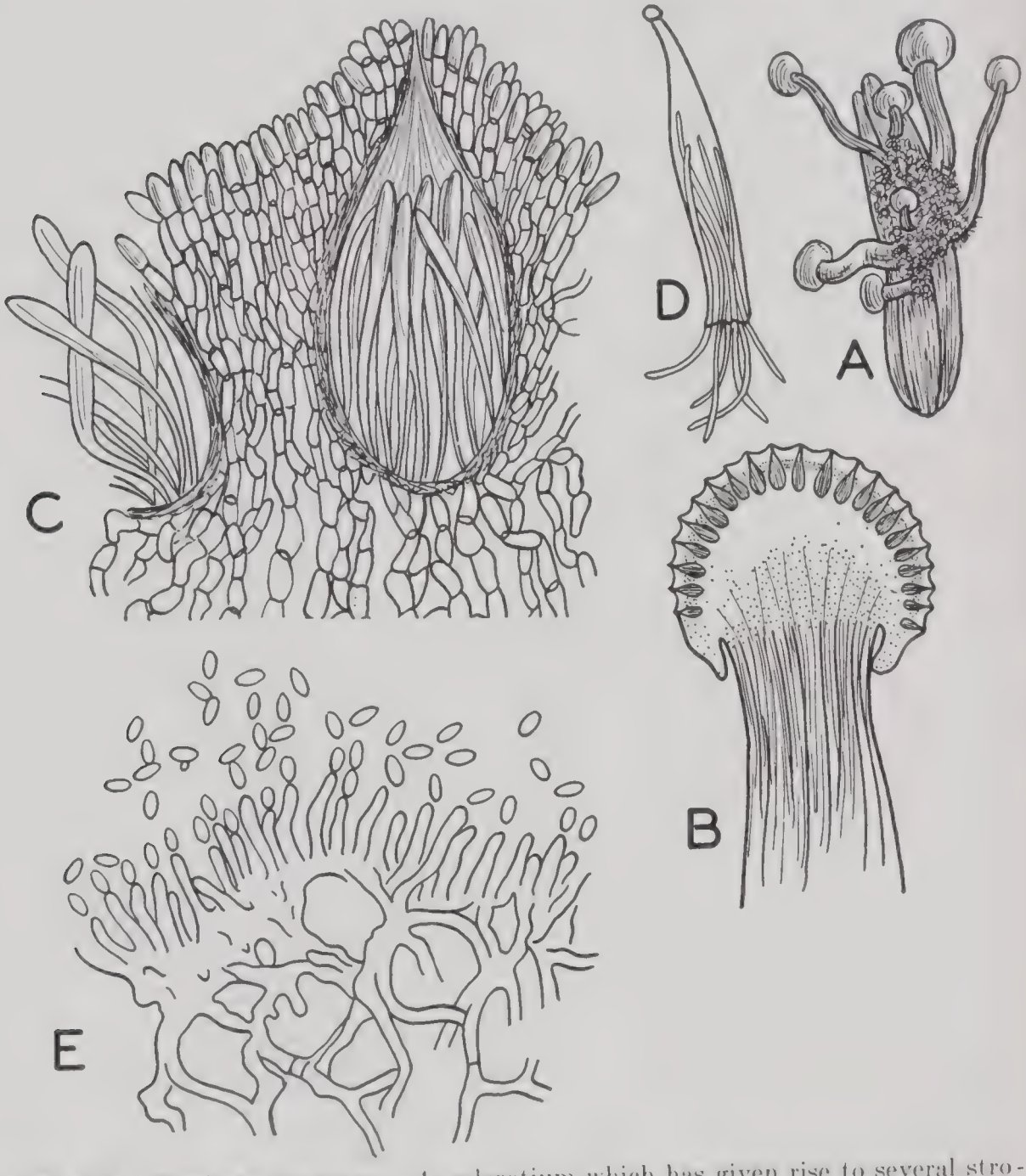

FIG. 103. *Claviceps purpurea*. *A*, sclerotium which has given rise to several stromata; *B*, median longitudinal section of stroma showing the perithecia; *C*, perithecium; *D*, ascus discharging ascospores; *E*, conidiophores and conidia. (*After Tulasne.*)

infection. The degree of uniformity of flowering and the rate at which the flowering period is completed are also important. The rate of flowering may differ with the variety and is influenced by growing conditions within any one variety. Krebs (7) reported that in winter rye increase

in distance between plants resulted in more infection, owing to the increase in tillering and consequent prolongation of the flowering period. Lewis (8) reported that rye flowers bloom in recurring intervals over an entire field. For 15 min. a large percentage of the heads have one to five flowers open; these close rapidly, and for about 45 min. almost no flowers are open. Cloudy weather and temperature changes influence the rhythm of bloom. In general, the period of greatest susceptibility is between 7 and 11 A.M.

After infection, the absorptive hyphae are established in the vascular system of the rachilla, and a rapidly developing extramatrical hyphal mass appears on the ovary. Conidia are formed promptly at this point, and secondary infection may follow on the same host species or, more often, on species which flower slightly later than the one currently infected. Oftentimes the infection of grasses in headlands may provide secondary inoculum at a time when the commercial grain crop is in the susceptible flowering period.

Sclerotia in rye and other cultivated grains drop to the ground or are harvested and disseminated with seed grain. On some grasses the sclerotia remain with the glumes attached to the rachilla. If awns are present on the glumes, the latter with the sclerotia may become attached to the coats of passing animals and become disseminated in that way (16). The ascospores were claimed by Stäger to ooze out on the slimy surface of the sphacelium and from there become disseminated by insects. Others have claimed forcible ejection of the spores. Possibly both methods prevail. In the latter method dissemination by wind is important. The conidia are disseminated chiefly by insects, many species having been reported as vectors.

Varietal Resistance and Physiological Races. Physiological races have been described by Krebs (7) and by Mastenbroek and Oort (10). There are numerous records of differences in the relative incidence of the disease on varieties and species. The higher incidence of ergot on rye than on other cultivated grains may be due in part to the fact that the flowers are open-pollinated while those of wheat, barley, and oats are largely self-pollinated. No resistant varieties of commercial importance have been developed.

Control. The use of ergot-free seed is the first requisite for control. Rotation of susceptible grains crops with legumes or other nonsusceptible hosts reduces the overwintering inoculum. If the ergot fungus has become endemic on native grasses, mowing of borders and headlands before the honeydew stage will reduce secondary inoculum. Sometimes ergot is most prevalent in the margins of fields because of inoculum from sclerotia and diseased grass plants in the borders. It may be advisable in such a case to harvest the outside of the field separately.

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BROWN ROT OF STONE FRUITS

In 1796 Persoon recorded a fungus giving rise to tufts of conidia in chains on decaying fruits of pear, plum, and peach. After first naming it *Torula fructigena*, he transferred it to *Monilia fructigena* in 1801. For 100 years this and closely related species were recorded in the conidial stage on stone and pome fruits in Europe, America, and other parts of the world. In 1818 Ehrenberg described a species, *Oidium laxum*, which is now regarded as the same fungus as that described on apricot by Bonorden in 1851 under the name *Monilia cinerea*.

In 1893 Schroeter transferred the two species *M. fructigena* and *M. cinerea* to the ascomycetous genus *Sclerotinia* without having seen the ascigerous stage. Woronin in 1900 was the first to demonstrate clearly that *M. fructigena* of Persoon and *M. cinerea* of Bonorden were distinct species. In 1902, J. B. S. Norton in Maryland discovered the apothecial

stage on rotted peach. He regarded it as the perfect stage of *S. fructigena*. In 1905 Aderhold and Ruhland observed the perfect stage of *S. fructigena* in Germany and also secured the ascigerous stage on material sent to them by Norton from a Kansas orchard. The latter they believed to be the perfect stage of *M. laxa* (*cinerea*) and named it *S. laxa*. There is some doubt whether they were correct in this interpretation, but Wormald in England in 1919 also reported a perfect stage of *M. cinerea* as *S. cinerea*. It was later determined by Roberts and Dunegan (21) that a collection of the brown-rot fungus of eastern United States made at Bethlehem, Pennsylvania, was described by Winter in Germany in 1883 as *Ciboria fructicola*. This collection is the first one in which the ascigerous stage of any of the three presently recognized brown-rot fungi was recorded.

In the large amount of literature which has accumulated since 1796, much confusion has arisen as to the identity of the species concerned in any given situation. It has now been established that two species, *S. fructigena* and *S. laxa*, occur in Europe. In the United States, east of the Continental Divide, *S. fructicola* is the prevailing species with *S. laxa* reported in northeastern Wisconsin (5) and in Michigan. In the Pacific coast region both *S. fructicola* and *S. laxa* occur. Up to the present writing *S. fructigena* has not been found in America, and *S. fructicola* has not been collected in Europe. *S. fructicola* is the prevalent species in Australia and New Zealand.

In 1928, Honey (13) proposed that those species of *Sclerotinia* in which the imperfect stage consists of monilioid conidia (*i.e.*, in chains) be transferred to a new genus, *Monilinia*, of which he made *M. fructicola* the type species. This was followed by Whetzel (25), who retained the genus *Sclerotinia* for certain species in which macroconidia are lacking and apothecia arise from well-defined sclerotia [compare *Sclerotinia sclerotiorum* (Lib.) DBy., p. 364].

In 1876 von Thümen in Germany was one of the first to recognize the economic importance of the disease incited by one or another of the organisms. In the last decade of the nineteenth century brown rot was very destructive in western Europe and the United States, and it continues to be a major disease on both continents as well as in Australia and New Zealand. In the United States it is most important on stone fruits, particularly peach, in the eastern and southeastern States and on peach, prune, sweet cherry, apricot, and almond in the Pacific coast states. It is of relatively minor importance in this country on pome fruits.

The history of the disease and of the pathogens is given in detail by Roberts and Dunegan (21) and by Harrison (9).

Symptoms. The first symptoms to appear in the spring are on the blossoms. This phase is the more pronounced where *S. laxa* is concerned.

Infected flowers turn brown and die promptly, the dead parts clinging to the pedicel for an indefinite period. The pathogen may advance through the pedicel to the twig on which cankers develop; or the twig may be girdled, the leaves wilting and dying but remaining intact. Initial infection of leaves may occur with *S. laxa*, but it is relatively rare. Cankers of considerable size on large branches have been described, particularly on peach, but Willison (27), working in the Niagara peninsula area, is of the opinion that while the brown-rot fungus (*S. fructicola*) may invade stems during midsummer, any extensive cankers are incited by secondary pathogens. Affected blossoms and twigs bear the gray sporulating masses of the respective brown-rot fungus in moist weather. Extrusion of gum occurs commonly on diseased organs.

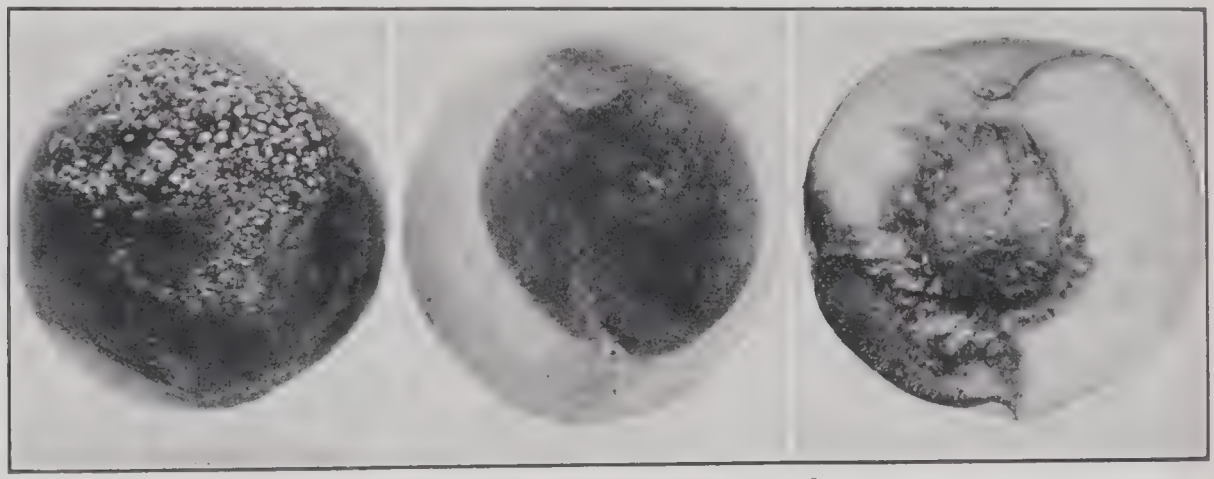

FIG. 104. Brown rot of peach.

Fruit rot is most severe on peach, but it may be very destructive on plum and cherry (Fig. 104). The entire peach crop may be almost completely destroyed on the tree within the period of a few days when environmental conditions are unusually favorable. Green fruits may be affected, but, in the main, fruit rot develops as the crop approaches maturity. In peach a tiny brown speck enlarges rapidly, commonly affecting the entire fruit in a few days. On the tree the rotted tissue is semiwatery, and on the fruit surface the gray mold develops abundantly in humid weather. Such tissue loses water fairly rapidly, and, on the tree, fruits dry down to mummies, which cling during the ensuing winter or drop to the ground.

On the Pacific coast where *S. laxa* and *S. fructicola* both occur, infection by the former results in relatively more severe blossom and twig blight, while the latter is more important as the incitant of fruit rot. In the United States brown rot is relatively rare and unimportant on apple and pear. In Europe where *S. laxa* and *S. fructigena* are the prevailing species, the

latter affects apple as well as stone fruits. In the latter host, blossom and twig blight as well as fruit rot occur.

The Causal Organisms.

1. *Sclerotinia fructigena* Aderh. & Ruhl., 1905.

Synonymy:

Conidial stage:

Torula fructigena Pers., 1796.
Monilia fructigena Pers., 1801.
Oidium fructigenum (Pers.) Kunze & Schmidt, 1817.
Acrosporium fructigenum (Pers.) Fr., 1822.
Oospora fructigena (Pers.) Wallr., 1833.
Oidium wallrothii v. Thüm., 1875.
Oidium fructigenum (Pers.) v. Thüm., 1879.
Sclerotinia fructigena (Pers.) Schroet., 1893.
Stromatinia fructigena (Pers.) Bos, 1903.

Ascigerous stage: *Monilinia fructigena* (Aderh. & Ruhl.) Honey, 1936.

2. *Sclerotinia laxa* Aderh. & Ruhl., 1905.

Synonymy:

Conidial stage:

Oidium laxum Ehr., 1818.
Acrosporium laxum (Ehr.) Pers., 1822.
Oospora laxa (Ehr.) Wallr., 1833.
Monilia cinerea Bon., 1851.
Monilia laxa (Ehr.) Sacc. & Vogl., 1866.
Sclerotinia cerasi Wor., 1888.
Sclerotinia cinerea (Bon.) Schroet., 1893.
Monilia oregonensis Barss & Posey, 1925.

Ascigerous stage:

Sclerotinia cinerea Wormald, 1921.
Sclerotinia cinerea f. *pruni* Wormald, 1921.
Monilinia laxa (Aderh. & Ruhl.) Honey, 1936.

3. *Sclerotinia fructicola* (Wint.) Rehm, 1906.

Synonymy:

Conidial stage: *Monilia cinerea* f. *americana* Wormald, 1919.

Ascigerous stage:

Ciboria fructicola Wint., 1883.
Sclerotinia cinerea f. *americana* Wormald, 1919.
Sclerotinia americana (Wormald) Norton & Ezekiel, 1924.
Monilinia fructicola (Wint.) Honey, 1928.

The morphological differences between the three species are not great. The conidia of *S. fructigena* are somewhat larger than those of the other two species, and en masse they are inclined to be yellow or buff in color rather than ashen gray. The ascospores of *S. fructigena* are sharply pointed at each end, while those of *S. fructicola* are rounded. The distinctions between *S. laxa* and *S. fructicola* are still less definite. The apothecial stage of the former is rare in the United States (Fig. 105). *S. laxa* forms mycelial cushions (sporodochia) on blighted parts in midwinter in the Pacific coast region and in early spring in Wisconsin. The cultural characters of the two species differ, but with the possibility of variation in either due to mutation, these characters cannot be relied upon too heavily

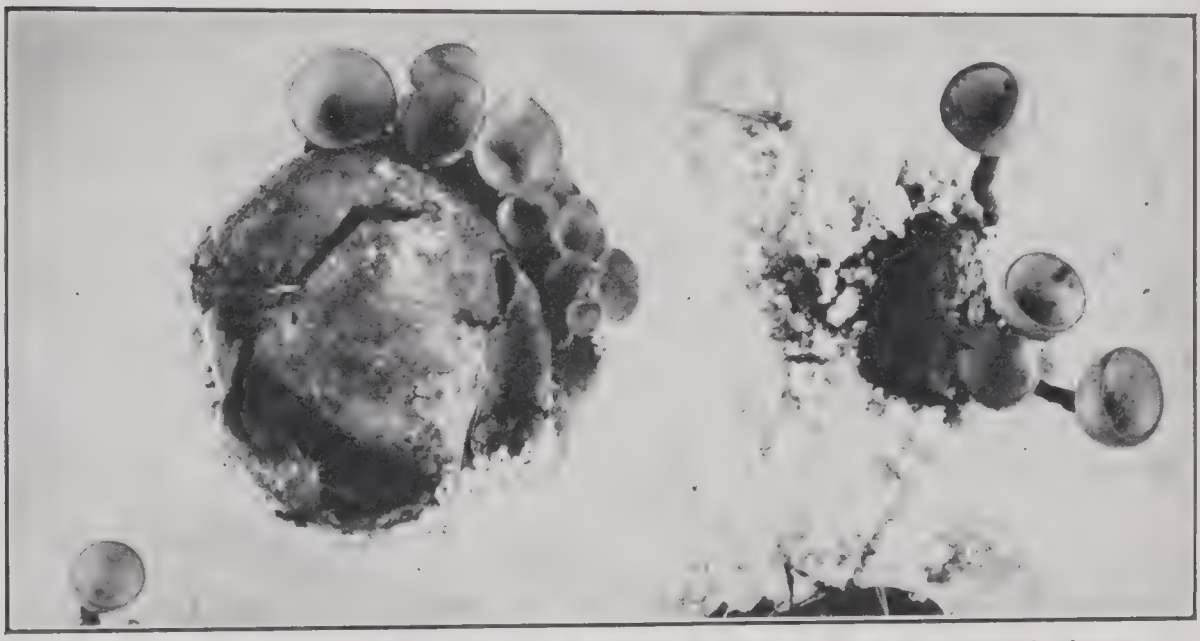

FIG. 105. *Sclerotinia fructicola*. Apothecia arising from mummied plums.

for purposes of diagnosis. The detailed description which follows refers to *S. fructicola:*

The mycelium is branched, hyaline, and multinucleate. Conidia are borne in branching chains on short, hyaline conidiophores, which occur commonly in tufts on the substrate; the spores are hyaline, ovate, continuous, and vary within the range of about 10 to 28 by 7 to 11 μ (Fig. 106). Microconidia are formed in old cultures and on fruit mummies; they develop in chains by the successive swelling of the tip of bottle-shaped sterigmata; they are hyaline, continuous, globose to subpyriform with a slightly concave base, 2.1 to 4.6 μ; they do not germinate. The function of the microconidia is not known, but similar bodies in certain other species of *Sclerotinia* are concerned with fertilization.

The fundaments of the sexual stage arise just below the surface of the pseudosclerotia which form in fruit mummies on or in the soil (11). Small,

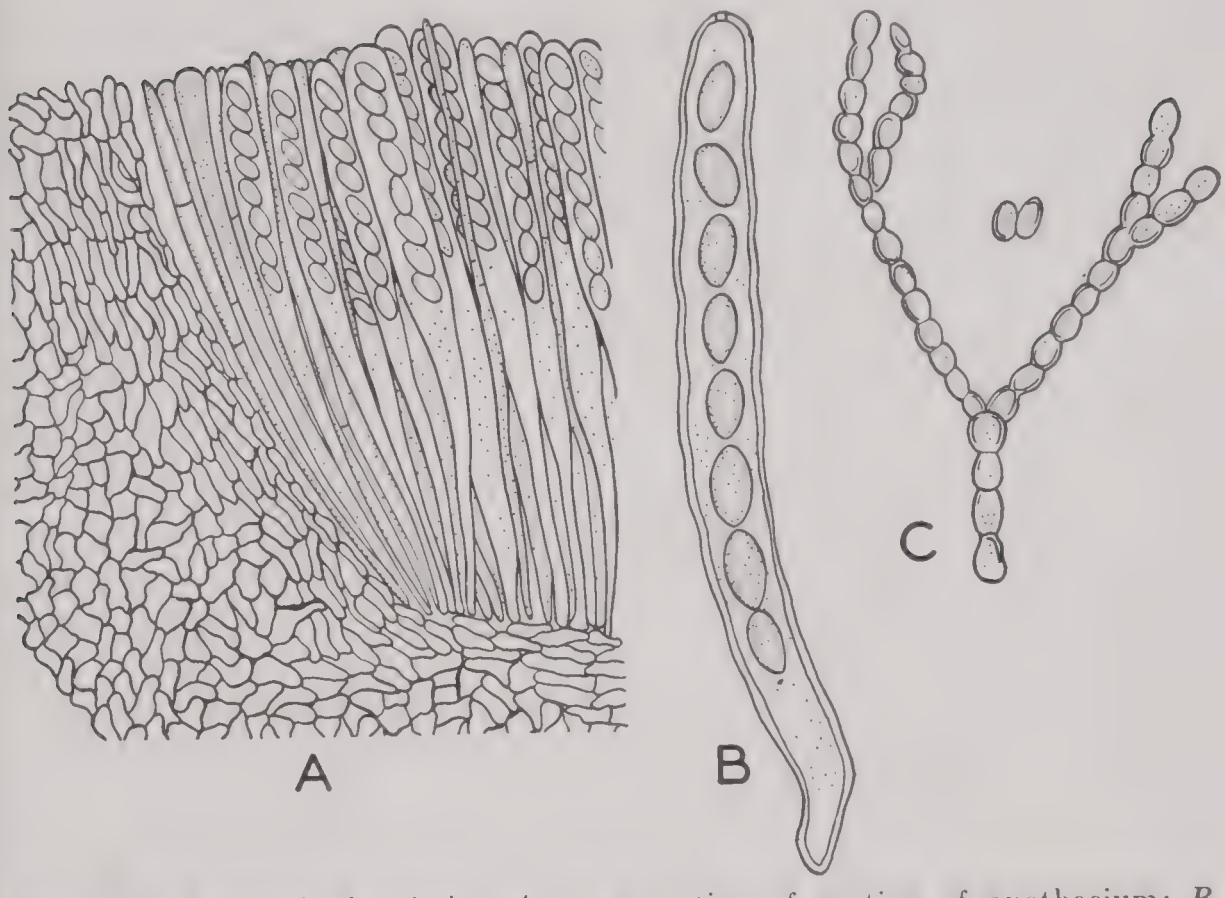

FIG. 106. *Sclerotinia fructicola*. A, cross section of portion of apothecium; *B*, ascus; *C*, conidiophore and conidia. (*A*, *B* after *Norton, Ezekiel, and Jehle; C after Cordley.*)

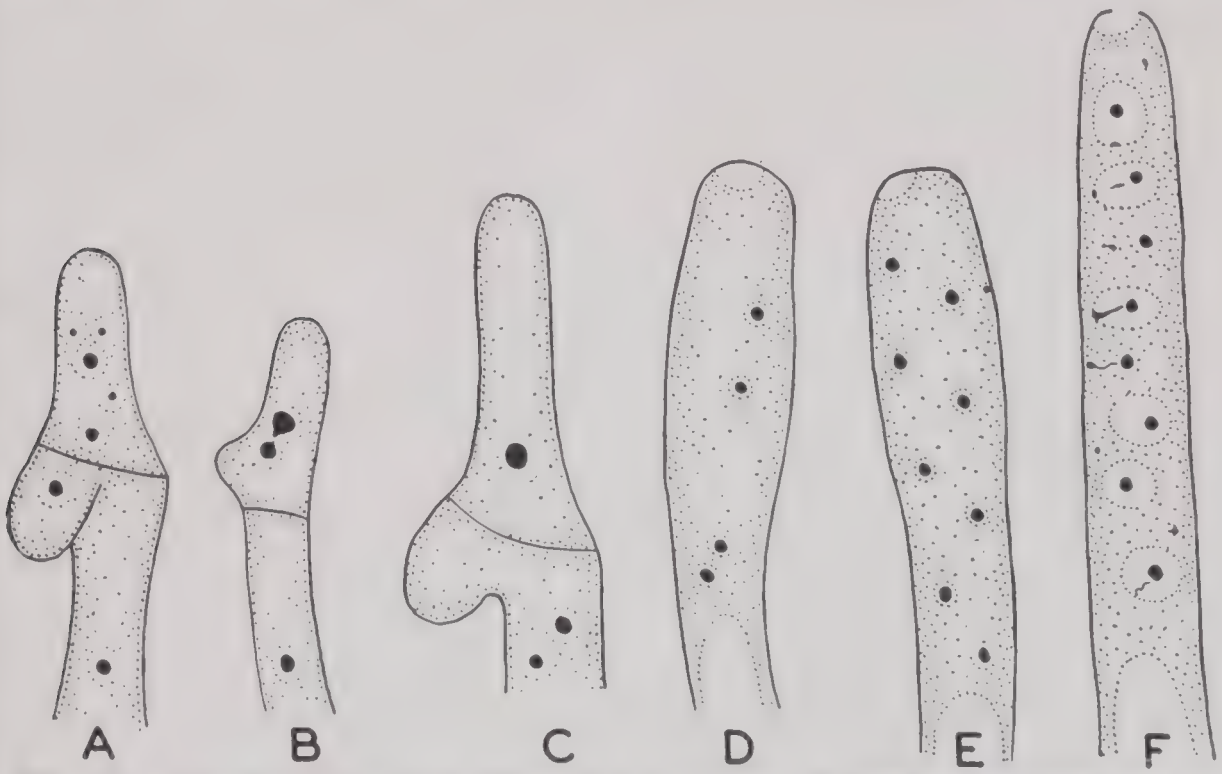

FIG. 107. *Sclerotinia fructicola*. Stages in formation of ascus. *A*, crosier before nuclear fusion; *B*, nuclei beginning to fuse; *C*, nuclear fusion completed; *D*, four-nucleate stage; *E*, eight-nucleate stage; *F*, delimitation of ascospores by free-cell formation. (*After Heuberger.*)

black, shiny, bulb-like dots appear and extend to form the stipe of the fruiting body. As the stipe grows, a depression appears at the tip which remains closed for a time and later opens to form the sporiferous cup. The stipe may be up to 3 cm. long, the length depending on the depth of the mummy in the soil. As the shiny, bulb-like dots appear, ascogenous hyphae may be detected in the subhymenium below the depression and where hyphae destined to become paraphyses prevail. The usual binucleate crosier cells appear; fusion of nuclei occurs; and asci push up between the paraphyses as they develop until they reach the same height as the latter. The fusion nucleus goes through three successive divisions and eight spores are formed (Fig. 107). The haploid number of chromosomes in the spores is four. The nucleus in each spore divides, making the mature ascospore binucleate. The ascospores are continuous, hyaline, and vary within the range of 6 to 17 by 3 to 8 μ. The fungus is homothallic.

Disease Cycle. The fungi overwinter in diseased fruit mummies on the tree, in twig cankers, and as pseudosclerotia in rotted fruits in or on the soil. The last means of overwintering leads to apothecial development in the spring by means of which ascospores become the primary inoculum. In the United States this holds chiefly for *S. fructicola* since the apothecial stage of *S. laxa* is relatively rare. In the areas where the latter species prevails, the major, if not exclusive, means of overwintering is in various locations on the tree, *i.e.*, twig cankers and diseased fruits. In California and in England, tufts of conidia are produced abundantly in midwinter when temperature and moisture become favorable. In some Pacific coast areas the fungi survive the dry summer period as dormant mycelium in blighted blossoms in which conidial production revives when humid weather prevails in the autumn.

Pseudosclerotia do not ordinarily produce apothecia if the mummies are buried more than an inch or two below the surface of the soil. They may produce fruiting bodies during the first spring, or they may not do so until the second spring. While it is known that some may be viable after several years, most of those which do not germinate disintegrate in the soil during the first 2 years. Soil moisture is the most critical factor in determining whether or not the apothecia form. They develop most effectively at 17 to 20°C.; they are retarded somewhat at low temperature and die rapidly at temperatures around 30°C.

After primary infection has occurred from conidia or ascospores, secondary spread is dependent upon air- and water-borne conidia. With *S. fructicola* a relative humidity of 85 per cent or above is required for conidial production. Conidia do not germinate readily in pure water, but when small amounts of available carbohydrates or other food materials

are present, they respond very promptly. In nature, such accessory products are supplied when infection drops settle on blossom parts or other plant organs. The optimal temperatures for germination of conidia and for growth of mycelium are about 25°C. High humidity is required for germination (6) and infection, and the minimum period within which the fungus penetrates varies from 18 hr. at 10° to 5 hr. at 25°.

Calavan and Keitt (5), in studies of *S. laxa* on cherry, found that most blossom infections originated through either the anthers or the stigma, although other blossom parts were attacked occasionally.

Curtis (7) studied penetration of numerous varieties of stone fruits by *S. fructicola*. She found that invasion might be directly through the cuticle or through stomata. The most usual method of effective entry in plums was through stomata; in peaches, down the hair socket; in nectarine, through the cuticle; in apricot, through cuticle or stoma. The fungus is intercellular in the early stages of disease development in peach fruits and largely so in plums (23). In general, ripe or nearly-ripe fruits are most susceptible, but earlier infection may occur through wounds. A common mode of spread from infected to noninfected immature fruits on the tree occurs where two such fruits are so situated as to be in close contact. Brooks and Cooley (3) studied infection of peaches after harvest by dusting conidia on wounded and unwounded peaches. Low temperatures during the incubation period had the greatest effect in reducing development of rot. This was confirmed later by Willison (28). The incubation period was longest when spores were dusted over unwounded fruits, shorter when dusted over fruits wounded by needle punctures, and shortest when spores were inserted in the wounds with the needle. Smith (22) showed that brushed peach fruits were infected the more rapidly because breaking of the hairs gave the germinating spores more ready access to the hair sockets.

Varietal Resistance. Differences in incidence of the disease on varieties of stone and pome fruits have been recorded (4, 7, 8, 10, 14, 17, 18, 23). Roberts and Dunegan (21) point out that in this country many of the older and often better flavored varieties of peach have been replaced by more resistant ones in commercial production. In the main, the resistant character is not sufficient to offer a major means of control under optimum environment for disease development and is expressed chiefly when fruits are immature. It becomes much less evident as the fruit approaches maturity and often disappears at the ripe-fruit stage. Valleau (23), at the University of Minnesota, studied plum varieties with reference to the basic factors underlying resistance and susceptibility. He found that those with the thickest and toughest skin were most resistant and that firmness of flesh at the ripe stage seemed to contribute something

toward resistance. Williaman *et al.* (26), working from the biochemical approach with materials from the same source, found that, in general, the more resistant varieties had higher crude-fiber content, a relation which held true particularly at the ripe-fruit stage. A correlation of higher pentosan content with resistance was observed, but it was not so marked as with the crude-fiber factor. Curtis (7) investigated the morphology of fruits in resistant and susceptible varieties of plum, peach, nectarine, and apricot. She found that the morphological characters which combined to afford some degree of resistance varied with the variety. In none was the fungus entirely excluded by cuticle or by plugging of the stoma. In Yellow Cherry plum, fruits of which were practically free of stomata, the cuticle was so thin in certain areas that penetration was not impeded, and the variety was one of the most susceptible. In other cases where the cuticle was quite thick and resistant, the number of stomata appeared to be more important than their structure. The hypoderm may play an effective part in resistance after penetration has occurred.

Control. Sanitary measures are obviously important. Fruit mummies should be removed from trees. Plowing of the orchard to bury mummies and eradication of wild *Prunus* are of value if practicable. Baur and Huber (1) tried various soil treatments to destroy apothecia in the spring but found calcium cyanamide the only effective material.

In Pacific coast areas and in Wisconsin and Michigan, where *S. laxa* prevails, dormant eradicant sprays are applied to reduce formation of sporodochia on blighted parts. Wilson (29) found calcium arsenite most effective for this purpose on apricots in California. Calavan and Keitt (5) had best results on sour cherry in Wisconsin with copper–lime–monocalcium arsenite plus fish oil, applied just before bud break; this was followed by 3–4–50 Bordeaux Mixture soon after blossoms had started to open.

Spraying procedures during the growing season vary with the region concerned and with the requirements for control of insects and of other disease incitants.

In Georgia early-season sprays have been found to be of little value except for insects, while midseason and preharvest sprays or dusts are of great importance in reducing postharvest rot. Self-boiled lime-sulfur was once used extensively on peach because of the sensitivity of this host to lime-sulfur, but wettable sulfurs are now substituted for the former. Carbamates were not effective on peaches in Delaware (20) but showed promise as preharvest sprays on cherry in New York (19).

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DROP AND WATERY SOFT ROT OF VEGETABLES

One of the most widespread and destructive diseases of vegetable crops is that known in its field aspects as drop and in storage and transit as watery soft rot. The first of three common pathogens, *Sclerotinia sclerotiorum*, was described in Europe in 1837; a second species, *S. minor*, was described in the United States in 1920; and a third, *S. intermedia*, in the same country in 1924. All three fungi incite similar diseases and all are world-wide in distribution. Ramsey (12) reported that more than 90 per cent of the isolates of *Sclerotinia* from decaying vegetables on the market over a 4-year period belonged to *S. sclerotiorum*.

Many vegetables are affected. Onion is an exception, but the neck-rot diseases have similar symptoms (p. 246). Potato and tomato plants are affected in the field, but rot of potato tubers is rare, and although tomato fruits are susceptible when inoculated artificially, natural occurrence of fruit rot is seldom seen. Severe cases of the disease on lemon (14), and on apricots in the green stage (15) have been reported in California.

While the fungi are widespread in occurrence in market and storage, they are most destructive in the field in the United States in the southern and Pacific coast regions, particularly on winter-grown vegetables. In the northern states the disease is found less often in the field, but it may be more prevalent on young plants in hotbeds and in greenhouse cultures of tomato, cucumber, and lettuce.

Symptoms. Growing plants are attacked at any time from the seedling stage to maturity. One of the characteristics of the disease on some hosts is that the fungus which infects the main stem somewhere near the soil line invades the cortical tissue rather rapidly without conspicuous effect until a sudden collapse of the plant follows. When the disease specimen is examined, superficial, white, cottony extramatrical mycelium is usually found to be fairly abundant, and young sclerotia may be present. The latter start as white compact bodies of hyphae up to the size of a barley kernel, varying in this respect with the species concerned. They are on the surface of the substrate or are partially embedded, and with age the outer layers of the sclerotia become jet black (Fig. 108). In some cases

the fungus invades the pith of growing plants and without producing the usual abundant extramatrical mycelium grows profusely and produces sclerotia within the pith cavity. This phase occurs commonly on potato, tomato, and cucumber and on seed plants of lettuce, cabbage, rutabaga, and turnip. In crucifer seed plants, the usual first evidence of disease is the formation of grayish linear lesions on the main stem beginning at the nodes (11). The final effect of pith infection is a slow stunting of the plant and premature ripening, rather than the sudden collapse (drop) which follows concentration of the pathogen in the cortex at the base of the plant.

FIG. 108. Watery soft rot of cabbage.

On mature storage organs the same type of mycelial and sclerotial development occurs. The host tissue becomes soft and watery as the disease progresses. The decayed portion of the organ loses water, sometimes rapidly, and finally desiccation follows. The effect on the host is not unlike that produced by the bacterial-soft-rot bacteria (p. 101), the chief diagnostic character of watery soft rot being the conspicuous mycelium in the early stages and the sclerotia in the later stages. A conspicuous and destructive phase, known as "nesting," commonly develops in the center of hampers of beans, peas, and other vegetables in transit, when the superficial mycelium grows very profusely on the substrate, binding the decayed material together in a cottony mass. The sclerotia of the watery-rot fungi are very similar in size, color, and shape to those of one or another

species of *Botrytis* (p. 246), and since the two groups of organisms have many hosts in common, they are often confused.

The Causal Organisms.

1. *Sclerotinia sclerotiorum* (Lib.) DBy., 1886.

SYNONYMY:

Peziza sclerotiorum Lib., 1837.
Peziza sclerotii Fckl., 1869.
Sclerotinia libertiana (Lib.) Fckl., 1869.

2. *Sclerotinia minor* Jagger, 1920.
3. *Sclerotinia intermedia* Ramsey, 1924.

The mycelium is hyaline and branched without any distinguishing features among the three species. While size of sclerotia varies somewhat with the environment, Ramsey (12) found those of each species to fall into distinct size groups. Under identical conditions the sclerotia of *S. sclerotiorum* ranged in diameter from 2.5 to 6 mm.; those of *S. intermedia*, 1 to 3 mm.; those of *S. minor*, 0.5 to 1 mm. Chivers (5) found that when *S. intermedia* and *S. minor* were grown at temperatures from 9 to 25°C., the sclerotia of the former were larger and those of the latter smaller as the temperature decreased. No definite period of dormancy appears to be necessary for the production of apothecia. Moist conditions are essential, and relatively cool temperature is most favorable. Light is necessary for full maturation; continued darkness results in abortion. The stipe arises from the sclerotium, ordinarily in the upper layers of the soil, and gives rise to a funnel-shaped cup from which ascospores are discharged in great abundance (Fig. 109). The ascospores are hyaline, continuous, and ovate. The average size of asci and ascospores, respectively, from various isolates of the three species follows: *S. sclerotiorum*, 8.2 to 10.2 by 125.4 to 160.4 and 5.9 to 7.3 by 11.7 to 15.1 μ; *S. intermedia*, 7.5 by 127.0 and 4.9 by 12.7 μ; *S. minor*, 8.9 by 141.0 and 7.0 by 14.1 μ (12).

No true conidia are produced. Microconidia are formed by each species, usually as the available food supply is on the decline. They occur in artificial culture, on sclerotia, and on the disks of overmature apothecia. They are formed in chains at the tips of short lateral branches of the vegetative mycelium; sometimes these sporiferous tips bear the spores endogenously within an old mycelial cell. They germinate very sparsely in water and culture media and apparently do not function in infection and in the dissemination of the fungi.

Disease Cycle. The fungus survives from one favorable crop period to another as sclerotia in the soil. The sclerotia are distributed on plant materials by implements, animals, and irrigation water (2) and with seed (12). Primary inoculum consists in part of mycelium arising from

sclerotia and affecting plants at or beneath the soil line but primarily of ascospores discharged from apothecia at the soil surface. Infection from ascospores may occur at any time the environment is favorable. Brown

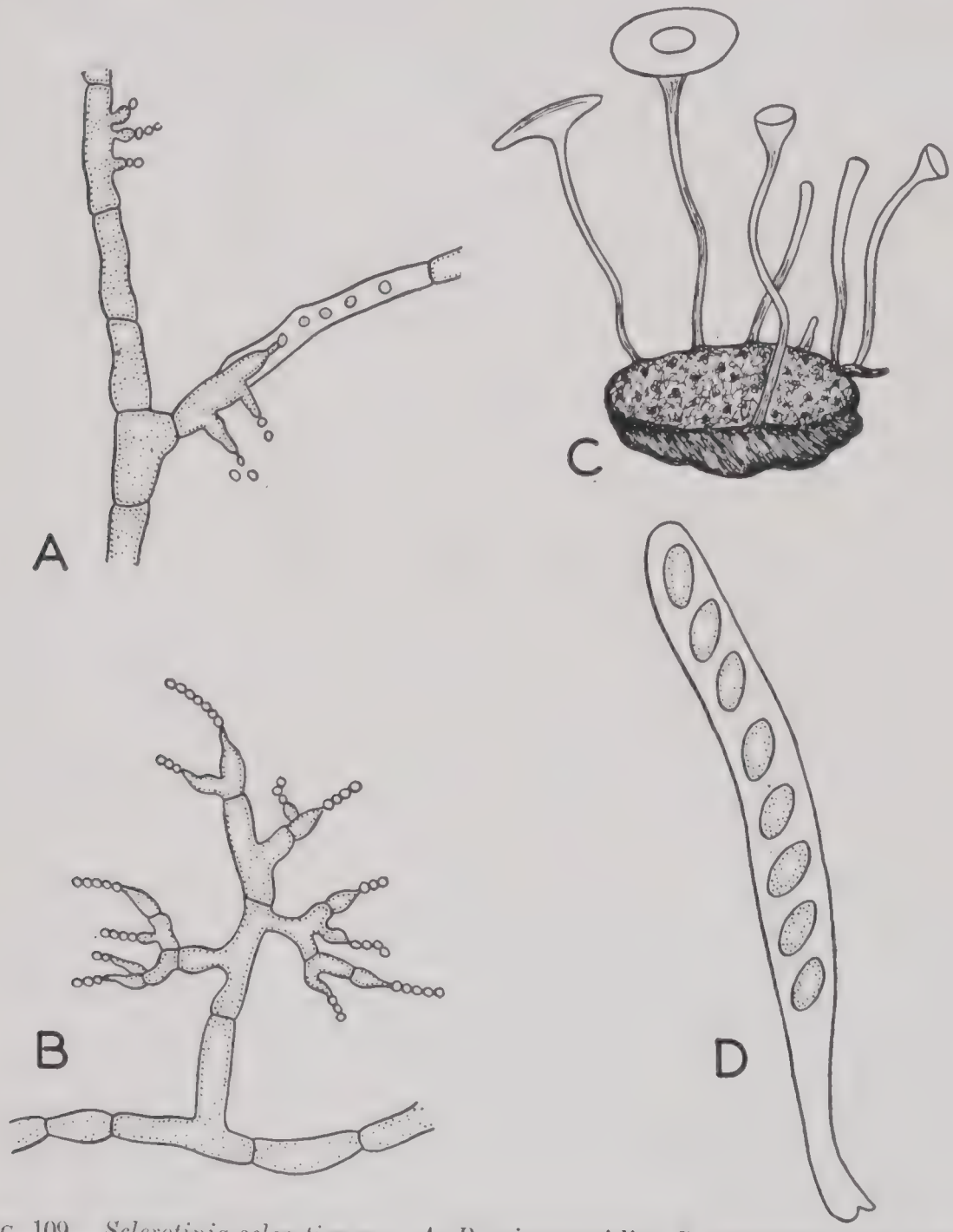

FIG. 109. *Sclerotinia sclerotiorum*. *A*, *B*, microconidia; *C*, apothecia arising from a sclerotium; *D*, ascus. (*A*, *B*, *D after Ramsey; C after De Bary.*)

and Butler (4) secured indications that ascospores were carried several miles by wind in a viable condition. The mycelium arising from the spores produces appressoria in contact with the host surface, and penetration is accomplished by mechanical rupture of the cuticle by a small in-

fection hypha (1). Once inside the cuticle, the fungus causes enzymic solution of cell walls in advance, and cells die some distance ahead of the invading hyphae. The work of De Bary (6) on the interaction of host and *S. sclerotiorum* is one of the pioneer studies of this type of parasitism. The more succulent plant parts are invaded much more readily and rapidly than tissues in which the cell walls have become suberized or lignified.

Apothecia are produced most effectively in moderately cool climate when soil moisture is adequate. The optimum temperature on water-agar substrate for stipe formation is 14°C. (7). The apothecia continue to mature, and mature fruiting bodies continue to discharge spores, over a considerable period under favorable environment. Since general infection, aside from that taking place in the soil, is dependent entirely upon continued production and dissemination of primary inoculum, epidemics are most common in areas of continuously cool moist weather. While no experimental evidence has been produced to explain why the disease is so uncommon in the north central and northeastern states even though the fungi have excellent means of surviving cold winters in the form of sclerotia, it is not unlikely that the basic explanation will be found in the lack of favorable environment for a protracted period of ascospore formation and dispersal, at a time when susceptible host plants are available.

S. sclerotiorum grows on agar over a range of 1 to 32°C. with the optimum at 24°. Infection of snap beans and carrots after harvest was found to occur over a range of 0 to 28° with an optimum of approximately 19 to 24° (8, 9). When the three species are compared under identical conditions, the rate of decay on most substrates is slowest with *S. sclerotiorum* and most rapid with *S. intermedia* (12). Ramsey (13), however, reported that when potato tubers were inoculated with each of the three species and held at 4° and 21°, decay was rare at both temperatures with *S. sclerotiorum;* it was pronounced at the low temperature and few infections occurred at the high temperature with *S. intermedia;* decay was rapid at the high temperature and absent at the low temperature with *S. minor.*

Control. The disease is one of the most difficult to control in the field. In areas where normal weather conditions favor apothecial development the key to control is suppression of the ascigerous stage, since ascospores are the only means of local dissemination. Rotation in intensive truck-crop areas is not very helpful because of long persistence of sclerotia and widespread dispersion of ascospores. Brooks (2) found deep plowing to be of little value, but some beneficial effects were derived by postharvest and preplanting applications of calcium cyanamide to destroy apothecia. Flooding of land during the summer reduced the incidence of viable sclerotia in Florida (10) and in Arizona (4). Brown (3) suggested pasturing sheep on harvested lettuce fields in Arizona and then quarantining animals

for 4 days, the maximum period for evacuation of viable sclerotia. In cold frames and greenhouses, soil treatment is the only feasible remedy.

Control in storage and transit is accomplished by attention to selection of stock that is as clean as possible. Although incipient invisible infection at this time may result in decay later, the disease can be held in check by precooling before loading for shipment and maintaining storage and transit temperatures as near 0°C. as is commensurate with the maintenance of good culinary quality.

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CHAPTER 9

DISEASES INCITED BY BASIDIOMYCETES

The Basidiomycetes comprise a diverse group of fungi including the smut fungi, the rust fungi, and most of the fleshy fungi. One common structure among the members of the class is the **basidium.** Typically it is a club-shaped or tubular organ which bears four basidiospores exogenously. The class is divided into two subclasses, the Heterobasidiomycetes and the Homobasidiomycetes. In the former are the smut fungi and the rust fungi; in the latter are the perfect stages of the *Rhizoctonia* fungi and essentially all of the wood-rotting shelf fungi.

While the basidium is the common organ which distinguishes the class, it varies widely in form. In the rust fungi, where it is known as the **promycelium,** it arises upon germination of the diploid teliospore, and during its development reduction occurs to form haploid nuclei. The basidium becomes septate to form uninucleate cells, the nuclei of which migrate into lateral buds which enlarge to become the **basidiospores,** or **sporidia.** In the smut fungi the diploid chlamydospore germinates to form a basidium (promycelium), which varies in size, shape, and septation among the species. Sometimes many haploid basidiospores (sporidia) are produced from a basidium; in other cases true basidiospores are absent. In the Homobasidiomycetes basidia vary in size and shape, and depending upon the species the spores are hyaline, pink, yellow, brown, or black. They are usually borne on slender protuberances from the basidium known as **sterigmata,** and the basidia are commonly arranged in a hymenial layer, which is often, but not always, part of a well-developed fleshy fruiting body.

The mycelium is usually branched and septate. Beginning with the basidiospore, which is usually uninucleate, the thallus is uninucleate at first. Eventually it becomes binucleate in a variety of ways, a process known as **dicaryotization,** or **diploidization.** The binucleate mycelium is referred to as the **dicaryotic** and the uninucleate mycelium as the **monocaryotic** stage. A comprehensive review of the literature on the **dicaryotization** process in Basidiomycetes and Ascomycetes is presented by Buller (3). He prefers the terms **diploidization, diploid,** and **haploid** over **dicaryotization, dicaryotic,** and **monocaryotic,** respectively.

While the dicaryotic stage is not strictly diploid in view of the fact that the two nuclei are haploid and often arise from different thalli (as in heterothallic species), it is distinctive in that the nuclei divide simultaneously. Such a pair of nuclei is known as a **dicaryon** or as **conjugate**

nuclei. The dicaryon is reproduced as the thallus grows throughout its vegetative phase, and the two nuclei fuse to form a truly diploid nucleus in the chlamydospore of the smut fungi, in the teliospore of the rust fungi, and in the basidium of the Homobasidiomycetes. Buller is of the opinion that the dicaryotic cell may properly be called diploid even though the two n genoms have not combined to form the $2n$ nucleus. It is best to refer to the thallus at this stage as $n + n$ rather than as $2n$. Numerous researches show that although the two genoms are in separate nuclei, their effect is the same as if they were combined in one $2n$ nucleus. The best evidence on this point is the fact that when one genom contains the dominant gene and the other genom the recessive gene of a gene pair, the dominant character (*e.g.*, color, growth habit, pathogenicity) is expressed in the dicaryotic tissue as fully as if the tissue were $2n$.

As in other classes of the fungi, a wide range of parasitism occurs in the Basidiomycetes. The rust fungi are all obligate parasites. The smut fungi are facultative saprophytes. Most of them can be grown on suitable culture media, but in only a few have chlamydospores been produced except upon the parasitized host. Many of the Homobasidiomycetes incite decay of mature wood, and many species of that subclass are obligate saprophytes.

Some of the major divisions of the Basidiomycetes and the positions of the pathogens treated in this chapter are presented below:

Heterobasidiomycetes.
- Ustilaginales. The smut fungi.
  - Ustilaginaceae.
    - *Ustilago maydis* (DC.) Cda., incitant of corn smut.
    - *Ustilago avenae* (Pers.) Rostr., incitant of black loose smut of oat.
    - *Ustilago kolleri* Wille, incitant of covered smut of oat.
    - *Ustilago nuda* (Jens.) Rostr., incitant of loose smut of barley.
  - Tilletiaceae.
    - *Tilletia caries* (DC.) Tul., and *T. foetida* (Wallr.) Liro, incitants of wheat bunt.
    - *Urocystis cepulae* Frost, incitant of onion smut.
- Uredinales. The plant rusts.
  - Pucciniaceae. Teliospores pedicellate or sessile but not united laterally, except in cases when they are borne on a compound stalk.
    - *Puccinia graminis* Pers., incitant of black stem rust of cereals.
    - *Puccinia malvacearum* Bert., incitant of hollyhock rust.
    - *Gymnosporangium juniperi-virginianae* Schw., incitant of apple and red cedar rust.
  - Melampsoraceae. Teliospores sessile, single or grouped, and embedded in the host tissue or joined together in layers or columns.

Melampsora lini (Pers.) Lév., incitant of flax rust.

Cronartium ribicola J. C. Fisch., incitant of blister rust of white pine.

Homobasidiomycetes.

Agaricales.

Thelephoraceae.

Pellicularia filamentosa (Pat.) Rogers., incitant of the common *Rhizoctonia* disease.

Helicobasidium purpureum (Tul.) Pat., incitant of violet root rot.

Polyporaceae. The polypores.

Fomes ignarius (L.) Gill, incitant of white heart rot of hardwood trees.

Agaricaceae. The gill fungi.

Armillaria mellea (Vahl.) Quel., incitant of mushroom root rot of orchard and other trees.

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CORN SMUT

Common corn smut, *Ustilago maydis* (DC.) Corda, is a disease which occurs widely and sometimes causes heavy damage. A second disease, head smut, *Sphacelotheca reiliana* (Kühn) Clint., is found chiefly in the western states but is the most prevalent of the two diseases on corn in Russia, India, and South Africa. The head-smut fungus also affects sorghum. The common corn-smut fungus affects only corn (*Zea mays* L.) and teosinte (*Euchlaena mexicana* Schrad.). Only the common smut will be considered here.

The amount of damage resulting from the disease is dependent upon the location of the gall. Garber and Hoover (8) found that damage was effected through increase in barrenness of diseased plants. Immer and Christensen (16) and Johnson and Christensen (18) showed that barrenness and reduction in yield were greatest when galls were located above the ear. Large galls on the tassel affected yield. Ears from smut-infected plants tended to have an inferior kernel luster. Jorgensen (20) showed reduction due to smut in yield of F_1 hybrids.

Symptoms. The most conspicuous phase of common corn smut is the production of galls on stems, leaves, axillary buds, ears, and parts of the male flowers (Fig. 110). The galls may reach the size of 4 in. or more in

diameter. As they begin to enlarge, they are light-colored, almost white. As the inner tissue darkens, owing to spore formation, the white outer membrane remains intact until a late stage, when it breaks and exposes the powdery black mass of spores. Depending upon the location of the gall, various morphological and physiological effects follow. Stem galls result in various degrees of yield reduction and sometimes in a bending of the stem. Infection through the silk results in smut galls in place of kernels. Galls on young seedlings may result in extreme dwarfing or death of the plant.

FIG. 110. Corn smut affecting the kernels of the ear. (*After Dickson.*)

The Causal Organism. *Ustilago maydis* (DC.) Cda., 1842.

SYNONYMY:

Lycoperdon zeae Beckm., 1768.
Uredo zeae Schw., 1822.
Ustilago zeae (Beckm.) Ung., 1836.
Ustilago schweinitzii Tul., 1847.
Ustilago zea-maydis Wint., 1881.
Ustilago euchlaenae Arcang., 1882.
Ustilago mays-zeae Magn., 1895.

The chlamydospores are spherical to ellipsoidal (8 to 11 μ), black, and heavily echinulated. Upon germination a septate promycelium is formed which bears ovate, continuous, hyaline sporidia (basidiospores) (Fig. 111). The sporidia bud profusely in yeast-like fashion. Considerable variation in germination is encountered in pedigreed lines of the fungus, *e.g.*, formation of sporidia directly from chlamydospores, formation of two opposite promycelia, branching of promycelia without formation of sporidia (23). The fungus grows well, vegetatively, on culture media, producing secondary sporidia in abundance as long as nutrients are sufficient. No chlamydospores have been described in culture. Fusion of sporidia in pairs is common, but there may be exceptions. According to Bowman (1) the nucleus in each sporidium divides and one nucleus from each spore migrates into the fusion tube, the latter being cut off by a septum, and from it a binucleate septate thallus arises. Sometimes the reduction division is delayed, giving rise to diploid mycelium in which reduction does not occur

until the next chlamydospores are formed (3). Occasionally binucleate sporidia are formed which give rise to binucleate mycelium directly (2). The fungus is largely intercellular after penetration, and chlamydospores are formed within the mycelial wall. In general, a haploid monosporidial line requires fusion with a compatible haploid line before typical invasion of the host tissue develops. Some solopathogenic monosporidial lines are found, however, which are diploid, owing to lack of meiosis during germination of the chlamydospore (3). Variants are frequent and numerous, hundreds of biotypes having been observed. These arise by mutation in haploid lines or by combination and segregation of genes through hybridization of compatible haploid lines. The rate of mutation is influenced

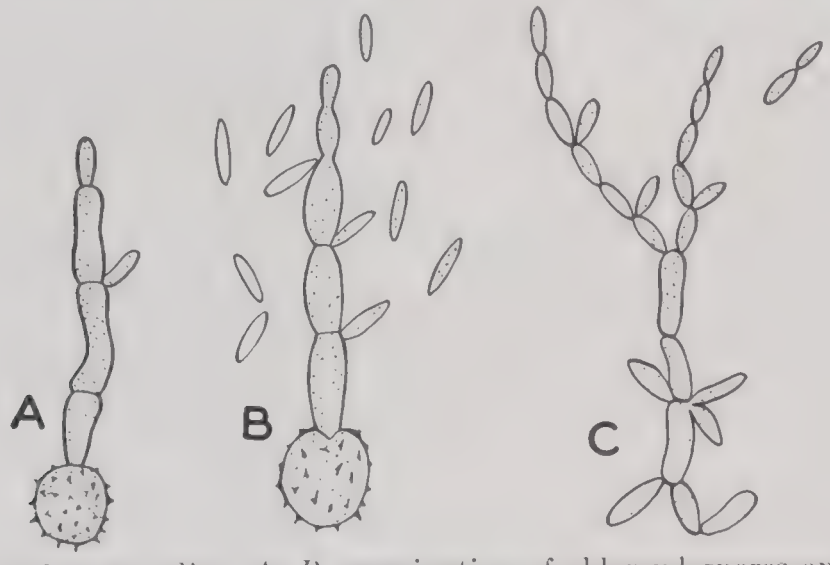

FIG. 111. *Ustilago maydis*. *A*, *B*, germination of chlamydospores and production of promycelium and sporidia; *C*, budding to form secondary sporidia. (*After Stakman.*)

by the substrate. The organism has been the subject of much fundamental study on variability in the fungi (3–5, 21–23, 27–31, 35).

Disease Cycle. While the chlamydospores may be carried on seed, the latter is not the major source of primary inoculum as is the case with many smut fungi pathogenic on small grains. The corn-smut fungus is uniquely adaptable as a saprophyte, and the chlamydospores persist on crop refuse and in the soil. Germination and sporidial production occur under favorable conditions on refuse and manure, and the primary inoculum comes from air- and water-borne sporidia. When young seedlings are infected, there may be systemic invasion of the plant, but most infections are local in nature. The embryonic tissues of all above-ground parts are susceptible to direct penetration (34). The disease would probably be more destructive if the vulnerable tissue just above the nodes were not ordinarily protected by sheaths consisting of older and more resistant tissue. The difference in apparent resistance between varieties is often based on the

morphological development of sheath and husk in relation to protection (25).

De Bary (7) in 1853 gave an account of the relation of the corn-smut fungus to the host tissue. He pointed out that the host cells enlarged (hypertrophy) and increased in number (hyperplasia) in response to invasion by the fungus. He noted that this process continued until spore formation began, at which time the protoplasm of the mycelium became concentrated in the sporiferous hyphae, while host-cell activity ceased and disintegration of the tissue followed. Knowles (24) reported hyperplasia and abnormal differentiation in xylem and phloem.

Varietal Resistance. The observation of differences in incidence of smut on varieties and breeding lines has been made by many investigators. In some cases the differences are in the nature of protective organs (25); in others it is based on incompatibility of the host tissue with the mycelium of the race or races involved (9). Owing to the extreme variability of the organism and the fact that most commercial varieties of the host are heterozygous open-pollinated lines or F_1 hybrids, the definition and cataloguing of races has not been carried out as in the rusts and in some of the small-grain smuts. Moreover, the variability of the organism has made for slow progress in incorporation of smut resistance in hybrid lines. Nevertheless distinct advances are being made, and hybrids with extreme susceptibility are avoided by breeders. In both sweet and dent corn many of the current hybrids possess substantial resistance to a major segment of the population of the corn-smut fungus (10 12, 15, 17, 19, 32).

Control. While sanitation and rotation might be expected to offer some control, they are not important measures in most cases because of the common intensive cultivation of corn combined with the fact that the sporidia are borne for considerable distances by wind.

The choice of the most resistant hybrid available with acceptable agronomic characters for the soil and climate concerned is the only feasible means of keeping corn smut in abeyance.

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OAT SMUTS

The two smut diseases of oat are called black loose smut and covered smut, respectively. They are world-wide in occurrence and cause considerable losses annually. Since the pathogens are quite similar in life history and since the two diseases occur commonly in the same field, they are considered herein collectively.

Symptoms. *Black Loose Smut.* The signs of the disease are confined to the panicle, where the individual flowers contain the black powdery masses of spores (sori) in place of the mature kernels. The sorus is covered temporarily with a delicate membrane which ruptures to expose the spores. Some variation in symptoms occurs with different races of the pathogen and varieties of the host (Fig. 112).

Covered Smut. The sori in covered smut are enclosed in a fairly permanent membrane of host tissue. The lemma and palea as well as the ovary may contain sori in some varieties (Fig. 112).

The Causal Organisms.

1. Black-loose-smut organism. *Ustilago avenae* (Pers.) Rostr., 1890.

Synonymy:

Uredo segetum var. *avenae* Pers., 1797.
Uredo segetum var. *decipiens* Wallr., 1815.
Ustilago avenae (Pers.) C. N. Jens., 1889.
Cintracti aavenae Ell. & Tracy, 1890.
Ustilago perennans Rostr., 1890.
Ustilago avenae f. *folliicola* Almeida, 1903.

The fungus is similar in morphology and life history to *U. nigra* Tapke, the incitant of semiloose smut of barley. The chlamydospores (6.5 by 7 μ) are dark brown to black with fine echinulations. On the septate promycelium are borne oblong to elongate, hyaline, uninucleate sporidia,

FIG. 112. Loose and covered smut of oat. (*After Dickson.*)

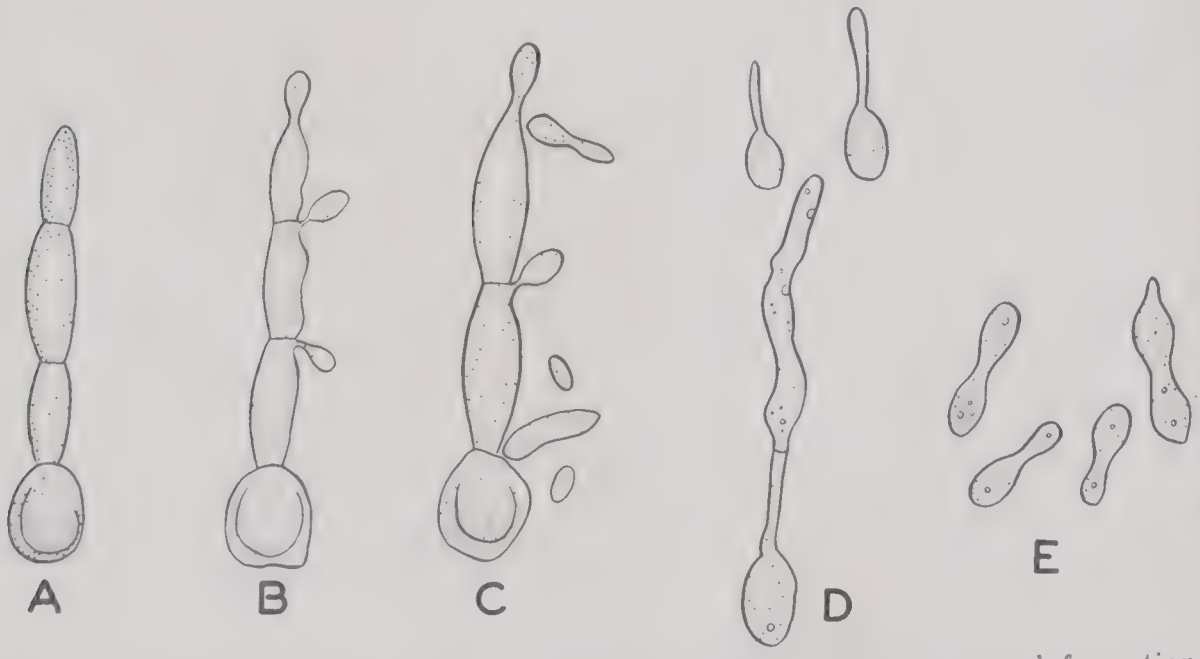

FIG. 113. *Ustilago avenae*. *A–C*, germination of chlamydospores and formation of sporidia; *D*, germination of sporidia; *E*, budding of sporidia. (*After Stakman.*)

which increase by budding and fuse in pairs to initiate the binucleate thallus. The fungus grows well on artificial medium (Fig. 113).

2. Covered-smut organism. *Ustilago kolleri* Wille, 1893.

Synonymy:
Ustilago avenae var. *levis* Kell. & Swing., 1890.
Ustilago levis Magn., 1894.

The fungus is similar in morphology and life history to *U. hordei* (Pers.) Lagerh. (p. 381). The chlamydospore is subspherical to angular (5 to 9 μ) with a smooth outer wall. It germinates to produce a four-celled promycelium and four ovate sporidia, which bud to form secondary sporidia. The fungus grows on artificial medium.

Holton (7) showed that the chlamydospore contains a single diploid nucleus. Reduction takes place during germination, and each cell of the promycelium contains one haploid nucleus. The sporidia of compatible haploid lines fuse in pairs, and the nucleus of one passes into the other to begin the dicaryotic phase. Although the nuclear history of the mycelium within the host has not been followed, it is presumed that it remains dicaryotic until chlamydospores are formed and fusion of haploid nuclei takes place in the maturing spore.

Fischer and Holton (6, 8) crossed *U. avenae* and *U. perennans* and studied the inheritance of the physiological properties responsible for characters of the sori, *i.e.*, naked and powdery, which were dominant over covered and indurate, respectively. They concluded that the two forms were races of the same species rather than distinct species. Fischer (5) recommended that for similar reasons *U. nigra*, incitant of semiloose smut of barley, also be placed in the species *U. avenae*.

Holton (7) discovered a buff-spored mutant from *U. kolleri* which he crossed with black-spore races of *U. kolleri* and *U. avenae*. The buff character is controlled by a single gene recessive to black. Five new buff races were produced through hybridization.

Disease Cycle. The primary inoculum consists largely of chlamydospores carried beneath the lemma and palea of the kernel. Falck in 1908 found occasional flower infection, but his discovery was not regarded as important until Zade in 1922 confirmed his observation (17). Dormant mycelium in the seed coat and spores within the floral bracts provide most of the primary inoculum. There is no overwintering in soil as in wheat bunt and onion smut. Infection takes place by binucleate hyphae in the early seedling stage. The hull of the oat kernel offers some mechanical protection as shown by the fact that spores applied to the external surface of the seed result in a lower percentage of infected plants than spores applied to dehulled seed. Since the spores may lodge beneath the hull, the effectiveness of some fungicides applied to the seed is reduced accord-

ingly. After penetration, the fungus is intracellular at first in mesocotyl, coleoptile, and first leaf. It then becomes intercellular, advancing with the growing point tissue and entering the flower primordia, where chlamydospores are formed. In varieties highly resistant to a given race, penetration occurs, but the fungus seldom advances to the culm primordia (18).

Reed and Faris (13) studied the relation of soil moisture, soil temperature, and soil reaction to infection by *U. kolleri*. Neutral to slightly acid reaction was most favorable. A distinct interaction of moisture and temperature was shown; at 15 per cent moisture, 15°C. was optimum; at 20 and 25 per cent moisture, 20° was best; and at still higher soil moisture the highest percentage of infection was at 25°. During emergence low moisture was more favorable than high moisture. With *U. avenae*, Bartholomew and Jones (1, 9) found 18 to 22° to be the optimum for infection, and high moisture was less favorable than low moisture.

Physiologic Races and Varietal Resistance. Reed (10) distinguished two physiologic races for each of the two species in 1924. Since that date some 30 races of *U. avenae* and 14 races of *U. kolleri* have been described. According to Dickson (4) Holton and Rodenhiser in 1946 differentiated 15 races of *U. avenae* and 7 of *U. kolleri*, by the use of 10 differential host varieties.

Reed (11, 12, 14, 15) also pioneered in the differentiation of varieties resistant to various races. Extensive breeding programs have been carried on in oats in which improvement of agronomic characters in combination with resistance to smuts, rusts, and other diseases has been the objective. A list of varieties resistant to many races of both smuts is given by Dickson (4). New varieties may be expected to appear which are adapted to various regions.

Control. Seed treatment is the first essential measure of control, even with resistant varieties, since there is always the possibility of an infective race and since treatment has value as a protectant against other pathogens in the soil. It is essential to use a volatile fungicide in order to reach spores lodged beneath the hull. Formaldehyde has long been used on oats, but New Improved Ceresan dust or Ceresan M have the advantages of ease of application, protective action, and less damage to seedlings (Chap. 16).

Smut-resistant varieties are now available for most localities, and they should be used generally.

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LOOSE SMUT OF BARLEY

Three smut diseases occur on barley. They are known as loose smut, *Ustilago nuda* (Jens.) Rostr., black semiloose smut, *Ustilago nigra* Tapke, and covered smut, *Ustilago hordei* (Pers.) Lagerh. The black-semiloose-smut fungus is closely related and similar in life history to the black-loose-smut organism pathogenic on oat (p. 377). The covered-smut fungus is, in like manner, similar to the covered-smut organism of oat (p. 377). The black semiloose smut and the covered smut of barley will not be treated in detail here. The loose smut of barley has symptoms which overlap those of black semiloose smut of barley, but the disease cycles are quite distinct. The barley-loose-smut organism is closely related and similar in life history to the wheat-loose-smut organism, *Ustilago tritici* (Pers.) Rostr.

While loose smut of wheat is not treated in detail in this book, it is well to point out that the disease cycle and the control of the disease on barley apply as well to wheat. The two loose smuts are distinctive among the smuts of small grains in that infection takes place in the floral organs of the growing plants and not in the young seedling following germination. The fungus lives over, therefore, as dormant mycelium in seed parts rather than as chlamydospores on the surface of kernels. Therefore seed treatment consists in seed disinfection rather than seed disinfestation, and hot water rather than chemical treatments are used in control (Chap. 16).

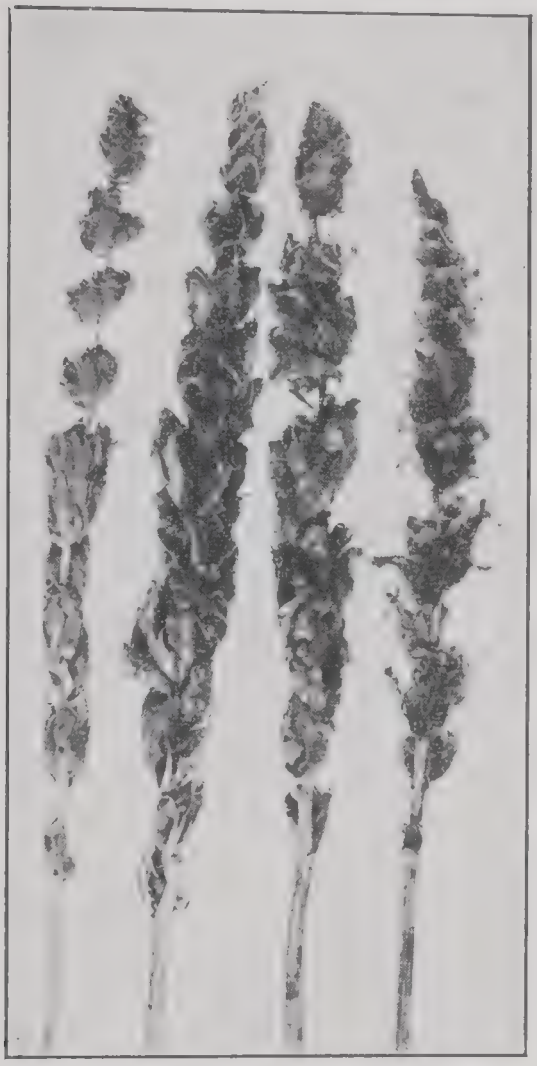

FIG. 114. Loose smut of barley. (*After Dickson.*)

Loose smut of barley is widespread in regions with relatively humid, cool weather during the flowering period. The fungus infects the three species of barley, as well as *Hordeum spontaneum* Koch and a number of species of grasses.

Symptoms. The spikes of infected plants emerge from the boot slightly in advance of those of healthy plants. The sorus occurs principally in the position of the seed, where it is covered at first with a membrane which ruptures early to expose the black powdery mass of spores (Fig. 114). Sori form occasionally in vigorously growing plants in leaves just below the spike. The spores are removed promptly by air currents at the time when normal plants are in the flowering stage. The bare rachises finally appear conspicuously above the level of the healthy mature spikes.

The Causal Organism. *Ustilago nuda* (Jens.) Rostr.

SYNONYMY:

Ustilago segetum var. *hordei* f. *nuda* C. N. Jens.
Ustilago segetum var. *nuda* C. N. Jens.
Ustilago hordei var. *nuda* C. N. Jens, 1889.
Ustilago nuda (C. N. Jens.) Kell. & Swing, 1890.

The chlamydospores (5 to 9 μ) are finely echinulate and dark-colored but slightly lighter on one side. Germination takes place by the formation

of a promycelium which has one to four cells (Fig. 115). No sporidia are produced, and under favorable nutrition, branching and segmentation may occur. The promycelial cells are haploid, and fusion between compatible cells takes place by conjugation tubes. Hyphae arising from conjugated cells are predominantly binucleate, and the infecting mycelium remains binucleate up to chlamydospore formation. Thren (14, 15) reported that at 1 to 2°C. the promycelial cells separate, the haploid cells segregating into two intercompatible groups. When compatible cells are mated, hyphal fusion occurs. The fungus grows well on some artificial media. Thren (16) reported that addition of vitamin B_1 to the medium is sometimes essential for growth.

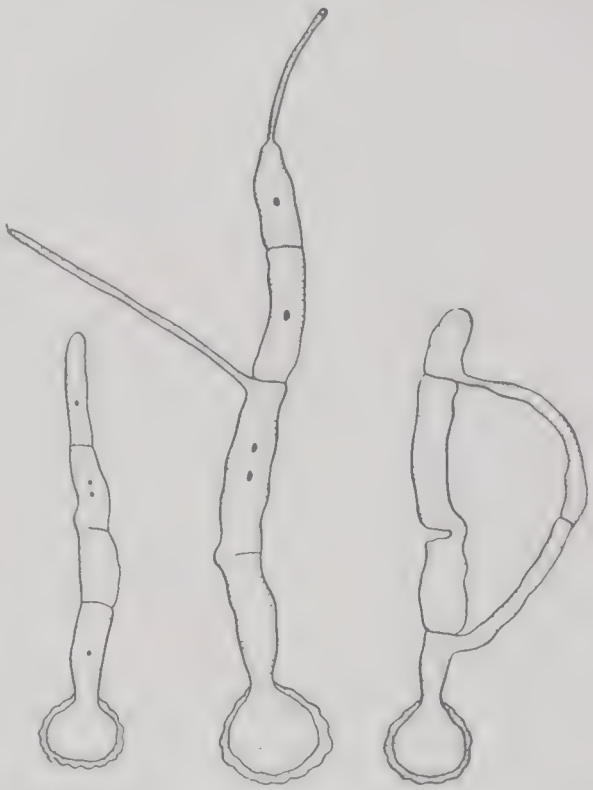

FIG. 115. *Ustilago nuda.* Germination of chlamydospores. (*After Dickson.*)

Disease Cycle. The chlamydospores from smutted spikes are carried by wind to healthy flowers, where they germinate, conjugate, and produce infective hyphae. Penetration occurs through the stigma or young ovary wall, the mycelium becoming established in the pericarp, in the remains of integuments, or in embryonic tissues before maturation. It remains dormant, chiefly in the scutellum (1, 11), until the following growing season. The chlamydospores are not long-lived as in most smut fungi. Humid weather at the flowering stage is essential for infection. The ovary and attachments become resistant to infection about a week after pollination.

When infected seeds germinate, the mycelium resumes growth, and development is not materially affected by environment. The fungus becomes systemic, and sporiferous branches appear in the mycelium in the spike, giving rise to chlamydospores. Earlier evidence (17) of infection through inoculation of the seed at planting time is now attributed to mixtures with the black-semiloose-smut fungus (12).

Physiologic Specialization and Varietal Resistance. Since the fungus tends to inbreed, cross-fertilization between strains is less common than in many species. Some physiologic specialization has been noted, however. Rodenhiser (7, 8) described differences in behavior in culture between collections from various parts of the world.

Many desirable smooth-awned varieties are susceptible to loose smut.

Resistant varieties occur (2–6, 9, 10, 13, 18, 19), and some are useful, chiefly as breeding parents. The inheritance of resistance has not been worked out fully, but it is apparent that several gene pairs governing resistance occur (2, 3). Shands (10) has shown a close linkage between genes for resistance to black stem rust (p. 401) and to loose smut. Schaller (9) has reported on the inheritance of resistance.

Control. As yet, loose-smut resistance has not been incorporated in varieties in agronomic use. Since chlamydospores are short-lived, emphasis in control is centered on seed treatment. Chemical disinfestants effective against other small-grain smut fungi, except that of loose smut of wheat, do not reach the fungus which has infected the barley kernel. The only satisfactory treatment is that with hot water. The seed should be presoaked in cold water for 12 hr., treated in water at 54°C. (129°F.) for exactly 13 min., cooled, and dried.

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BUNT, OR STINKING SMUT, OF WHEAT

In Chap. 2 on History of Plant Pathology wheat bunt is frequently mentioned, since it was one of the most severe diseases in the Middle Ages and was the subject of observation and study by many writers as early as Theophrastus. Tillet was the first to make distinction between bunt and loose smut, and he established the infectious nature of the former disease. Prévost in 1807 published his classical paper which dealt extensively with bunt. This was the first case in which the pathogenic relation of a microorganism as a causal factor in disease was established. The Tulasnes, De Bary, Kühn, and Brefeld were all students of bunt, and each contributed to the basic facts concerning it. Literature reviews have been compiled by Woolman and Humphrey (38) and by Holton and Heald (21).

This world-wide disease occurs throughout the wheat-growing regions of the United States. Until brought under control by seed treatment and the use of resistant varieties, it was very destructive in many areas, particularly in the Palouse region of Washington, Idaho, and Oregon. Two distinct species of pathogen (*Tilletia caries* and *T. foetida*) incite the disease. *T. foetida* is the more common in north central and eastern states while *T. caries* is more abundant in the western states. They infect rye occasionally, and some of the species of *Aegilops*, *Lolium*, *Agropyron*, and *Hordeum* are hosts of one or another of them. They have been shown to infect both bread wheat, *Triticum vulgare*, and durum wheat, *T. durum*, and 11 other species of *Triticum*.

An obnoxious odor given off by the spores of the bunt fungi and resembling that of decaying fish is the basis of the common term stinking smut. The odor is due to trimethylamine. Some races of *T. caries* and *T. foetida* do not produce the volatile material, and with those which do produce it the host variety has some effect on the amount. The damage caused by bunt is expressed in various ways. Reductions in yield and in the quality of the threshed grain are most important. Another damage, now generally corrected, is that to harvesting and threshing equipment due to

explosions instigated by the concentration of smut spores in the atmosphere. The allergic effect of the spore dust on man during handling of smutted grain is also a major factor.

Symptoms. The signs of the disease are usually not evident until the plant is in the heading stage. Both *T. caries* and *T. foetida* have an effect upon the physiology and growth of the host. While many observations have been made in considerable detail as to the effect of each species, it is evident now that races within a species may have quite different influences upon host response. Stunting of growth is common. Dwarf

FIG. 116. Wheat bunt in two varieties of wheat. In each case the healthy head is on the left and the diseased one on the right. Healthy kernels above, diseased kernels below. (*After Dickson.*)

bunt is the name applied to the disease incited by a race of *T. caries* in which pronounced stunting of the host occurs. Abnormal morphology of the spike, difference in development of floral parts, and abnormal color of ovaries are some of the striking effects (Fig. 116). Increase in tillering is not uncommon, but the opposite effect has also been observed. Reduction in root development was observed by Tillet and has been confirmed by others. Increased susceptibility to stripe rust, *Puccinia glumarum* (Schum.) Eriks. & E. Henn., and to *Fusarium* seedling blight and increased resistance to powdery mildew have been recorded.

Some of the common signs of bunt are described by Barrus (2), but it is well to bear in mind that variation in host, pathogen, and environment

modify the symptoms greatly. The pistil is enlarged and the ovary is longer and broader than normal. The latter organ tends to take on an abnormally green color. Stamens are reduced in length, and anthers are more yellowish than normal. The diseased heads are usually slim compared with healthy ones, and they retain their greenish cast longer. The diseased kernels are off-color, tending to be grayish brown in contrast to the golden-yellow or red of healthy kernels. The final diagnostic sign is the distinctive black powdery mass of spores, which is seen to occupy the entire kernel within the pericarp.

The Causal Organisms.

1. *Tilletia caries* (DC.) Tul., 1847.

Synonymy:

Lycoperdon tritici Bjerk., 1775.
Uredo caries DC., 1815.
Uredo sitophila Ditm., 1816.
Tilletia sitophila (Ditm.) Schroet., 1877.
Tilletia tritici (Bjerk.) Wint., 1884.

2. *Tilletia foetida* (Wallr.) Liro, 1920.

Synonymy:

Erysibe foetida Wallr., 1833.
Ustilago foetens Berk. & Curt., 1860.
Tilletia levis Kühn, 1873.
Tilletia foetans (Berk. & Curt.) Schroet., 1877.

The life histories of the two species are quite similar. One of the chief morphological distinctions is in the markings of the outer wall of the chlamydospore. In *T. caries* the wall has reticulations ranging from minute shallow meshes to deep indentations; in *T. foetida*, the walls are smooth. The chlamydospores of *T. caries* are 15 to 23 μ in diameter; those of *T. foetida* are 17 to 20 by 18 to 22 μ. Sterile cells, common among the functional chlamydospores, are thin-walled, hyaline, and slightly smaller than the latter. The chlamydospores are various shades of brown and are black en masse. Hanna (14) and Holton (17) crossed *T. caries* and *T. foetida* and described hybrid races. The chlamydospores germinate by production of promycelia (basidia) at the apex of which are produced filiform, hyaline, primary sporidia, 8 to 16 in number, which fuse in pairs *in situ* to form characteristic H-shaped structures. In the dwarf strain of *T. caries* the promycelium forms branches on which sporidia form, some of them fusing. The fused primary sporidia germinate, and on the mycelium are borne sickle-shaped, hyaline, secondary sporidia (Fig. 117).

Rawitscher (31) reported in 1914 that the promycelium of *T. caries*

and *T. foetida* contains usually eight nuclei, which he considered to arise from the diploid nucleus in the chlamydospores. Each nucleus migrates into a sporidium, and after the fusion of sporidia in pairs, nuclei migrate into the hyphal branch developing from the pair. The mycelium and secondary sporidia are binucleate, and infection occurs only by the binucleate thallus. The young chlamydospore is binucleate, the nuclei fusing in the mature chlamydospore. There is a difference of opinion among investigators as to whether the diploid nucleus divides in the chlamydospore or in the promycelium, whether or not the latter becomes septate, and whether the binucleate state always begins with fusion of sporidia. Buller and Vanterpool (8) distinguished between uninucleate and binucleate secondary sporidia.

FIG. 117. *Tilletia caries*. *A*, germination of chlamydospore; *B*, fusion of primary sporidia; *C*, formation of secondary sporidia. (*After De Bary.*)

Brefeld (4) was the first to grow *Tilletia* in nutrient solution. Sartoris (33) succeeded in growing the fungus on solid medium. Flor (12) secured monosporidial lines by isolating the primary sporidia with a glass needle. Secondary sporidia are produced abundantly in culture. He inoculated by mixing compatible lines and inserting inoculum into the base of the seedlings. Churchward secured no penetration by mycelium from fused primary sporidia or from single secondary sporidia and concluded that hyphal fusion was a prerequisite of invasion of the host (9).

Disease Cycle. Primary inoculum consists of spores carried on seed or present in the soil. Spores have been shown to remain dormant in the soil for several years. The hypha produced by fused primary or secondary sporidia forms an appressorium in contact with the cuticle, and a penetration tube invades intercellularly. When it reaches the culm primordium, it grows with the host tissue until heads are being produced (9). The chlamydospores then begin to mature in the internal tissue of the kernel. It has been shown by Milan (28, 29), by artificial inoculation under special conditions, that infection may occur on older plants by injection through needle pricks at the base of the young culm.

Early workers indicated that temperature had an effect upon germination of chlamydospores. While many observations have been made, there

is as yet no general agreement on the matter. Woolman and Humphrey (39) stated the cardinal temperatures to be minimum, 0 to 1°C.; optimum, 18 to 20°; maximum, 25 to 29.1°. Hahne (21) in Germany reported 4°, 18 to 20°, and 36°. Bressman and Hanna (21) noted that physiologic races differ in the relation of temperature to germination. Lowther (27) found the dwarf-bunt race of *T. caries* to have an optimum at 5° and an apparent maximum at 10°. As early as the seventeenth century it was noticed that temperature and moisture at and subsequent to sowing wheat were important in relation to development of bunt (38). Hungerford (24) in 1922 showed that low soil temperature and high soil moisture are conducive to infection. The optimum soil temperature was 9 to 12°C. Faris (11) also secured best infection at relatively low temperatures. Johnston (25) in Kansas secured progressively higher percentages of infection in successive plantings of winter wheat from Sept. 15 to Nov. 1. These observations have been confirmed in various parts of the world, but it has also been shown that various other factors interact with temperature and moisture; these factors include soil type, soil fertility, host variety and physiologic race, spore load, depth and rate of seeding, and length of day (21). Many resistant varieties are more susceptible at low than at higher temperatures.

Physiologic Specialization and Varietal Resistance. The first evidence of physiologic races in *T. caries* and *T. foetida* was published by Faris (11) in 1924. Since that time many investigators in various parts of the world have studied this subject and reported distinct races. Holton and Heald (21) in 1941 stated that at least 139 races had been reported, but they pointed out that there was much duplication because of a lack of a uniform system of classification. They have reviewed and summarized the literature on the subject up to 1941. Flor (12) in 1931 and 1932 demonstrated heterothallism in *T. caries* and *T. foetida*. Hanna (14) obtained hybrids between *T. caries* and *T. foetida*. Holton (19) derived a new race by hybridizing two races of *T. caries*. The factors for pathogenicity are controlled by genes inherited on a multiple-factor basis, which are independent of those controlling spore morphology (17).

Differences in varietal susceptibility to bunt have been observed for many years. Farrar in Australia was the first to breed wheat systematically for bunt resistance, beginning about 1900. Woolman (37) showed that the fungus penetrated both susceptible and highly resistant varieties with equal facility, but it failed to develop beyond entrance of the epidermal cell in the latter. Gaines (13) in 1920 published on the inheritance of resistance to smut. A vast amount of investigation on genetics of smut resistance and on the development of resistant varieties has continued in various parts of the world (5, 23, 35). Much of the earlier work is confus-

ing because of the various criteria used in classifying resistant and susceptible plants and because of differences in the number and identity of races used. Briggs and associates (6, 7, 26, 34) defined six genes responsible for resistance to a single race of *T. caries*. Resistant varieties are not necessarily universally and permanently so, because of variation in population of races and the possibility of the appearance of new or unknown races. Many satisfactory varieties are now in use, and continual replacements as a result of breeding programs are to be expected.

Control. The early development of seed treatment centered around the control of bunt. The early history of the subject is discussed in Chap. 16. Present-day control centers around three major lines of approach: (*a*) cultural practices, *e.g.*, rotation, time of sowing; (*b*) seed treatment; (*c*) use of resistant varieties, as discussed in the previous paragraph.

In general the first of these is not used except in special cases because of incompatibility with other farm practices (15, 16, 21).

Seed treatment is widely practiced, and an immense amount of research on fungicides for this purpose has been carried out. The copper carbonate dust, New Improved Ceresan, and Ceresan M (slurry) are the most widely used (Chap. 16). As yet, no satisfactory means of protecting seedlings from spores in the soil has been devised. It is, therefore, necessary to supplement seed treatment by use of the most resistant, agronomically satisfactory variety adapted to the area concerned.

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ONION SMUT

Onion smut was first reported in 1869 in the Connecticut River valley, where by 1888 it was of great economic importance on old onion soils. It has become an important disease in a majority of the onion-growing areas throughout the northern states as far west as Oregon. The pathogen was undoubtedly introduced from Europe, where its occurrence was not recorded until some time later than the American record. A specimen of the fungus in the Persoon herbarium in France, collected about 1834, leaves no doubt as to its European origin. The disease has become important in numerous European countries and has been recorded from the Caspian Sea region, which is not far removed from the original habitat of the onion. It also occurs in New Zealand and Canada. In the United States the disease has remained strictly a northern one, not being reported south of Kentucky, although there is good reason to believe that the fungus has been transported frequently to southern growing regions. There is a similar concentration of the fungus in northern Europe in contrast to its very rare occurrence in or its absence from Spain, southern France, and Italy. Distribution in relation to environment is discussed under Disease Cycle.

The fungus is restricted to the genus *Allium*, within which there are a considerable number of susceptible species (3). Common economic hosts, besides onion, are leek (*A. porrum* L.) and Welsh onion (*A. fistulosum* L.).

Symptoms. The disease appears first on the cotyledon of the young plant soon after it emerges from the soil. The lesion consists of a dark, slightly thickened area involving leaf or cotyledon for one to several millimeters. Sometimes the major part of a leaf is taken up by a single lesion, and in such cases the leaf tends to curve downward abnormally. On older plants numerous raised blisters occur near the base of the bulb leaves.

The lesions in plants at all stages often rift to expose a black powdery mass of spores (Fig. 118).

The fungus progresses inwardly from leaf to leaf at the base of the plant. The majority of infected plants die within 3 to 4 weeks after emergence. Some plants survive in a weakened condition until midseason or later, and occasional plants produce bulbs which bear the lesions on the outer fleshy

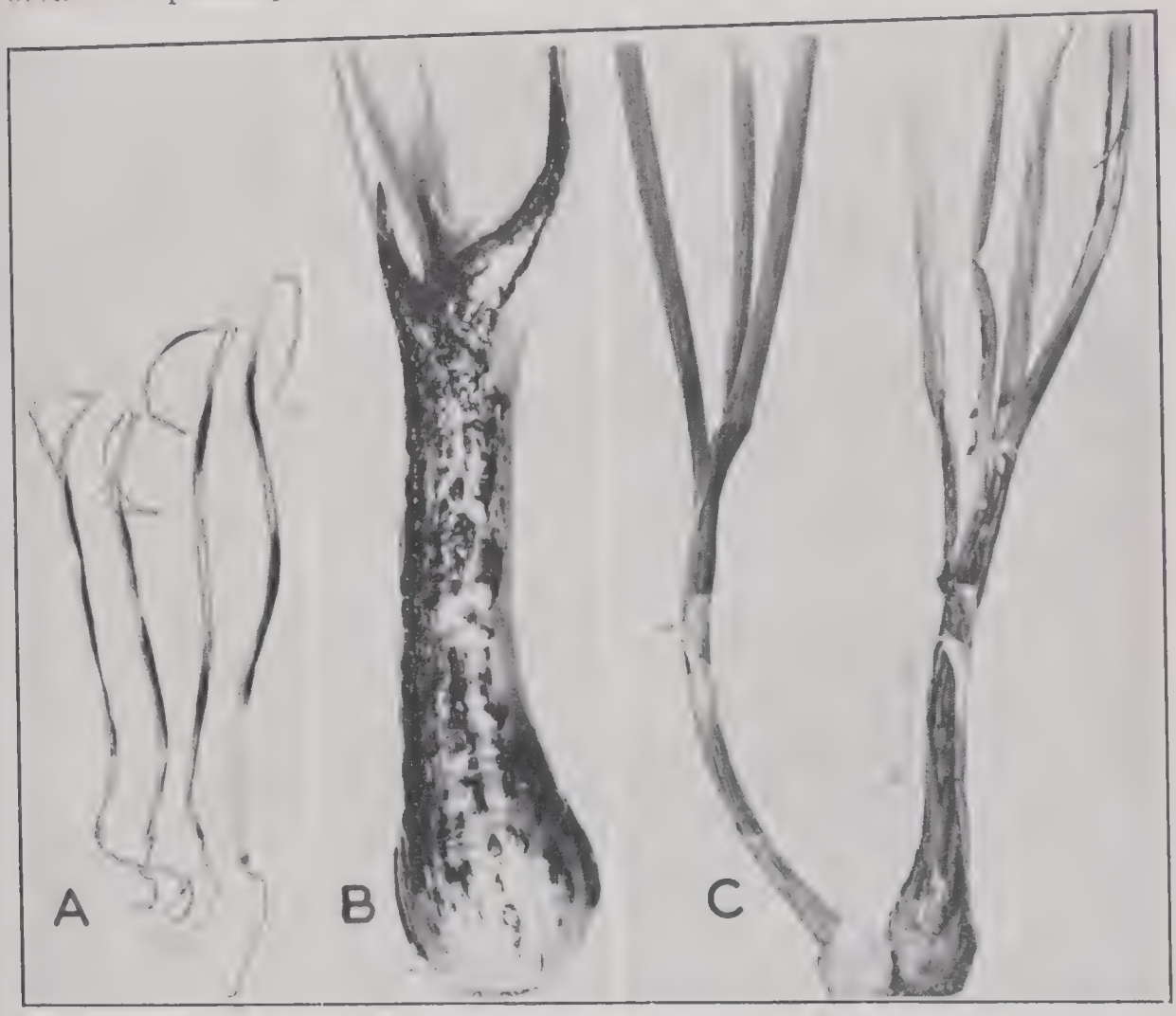

FIG. 118. Onion smut. *A*, lesions on young seedlings; *B*, lesions on half-grown plant; *C*, plants grown from a single infected bulb. One plant became systemically infected; the other remained free from infection.

scales and in one or more underlying scales. The fungus does not produce a rot in storage, but affected bulbs may be more subject than healthy ones to invasion by other pathogens. Very rarely, infected leaves may arise from a bulb which became infected during the previous season.

The Causal Organism. *Urocystis cepulae* Frost, 1877.

SYNONYMY: *Tuburcinia cepulae* (Frost) Liro.

The chlamydospores consist of a single dark-colored, thick-walled central cell (rarely two such cells exist) to which is attached a series of smaller,

hyaline, thinner walled appendage cells. This type of compound spore is characteristic of the genus *Urocystis*. The central spore is 12 to 15 μ in diameter. The germination of the chlamydospores of species of *Urocystis* usually consists of a promycelium with apical sporidia. It was

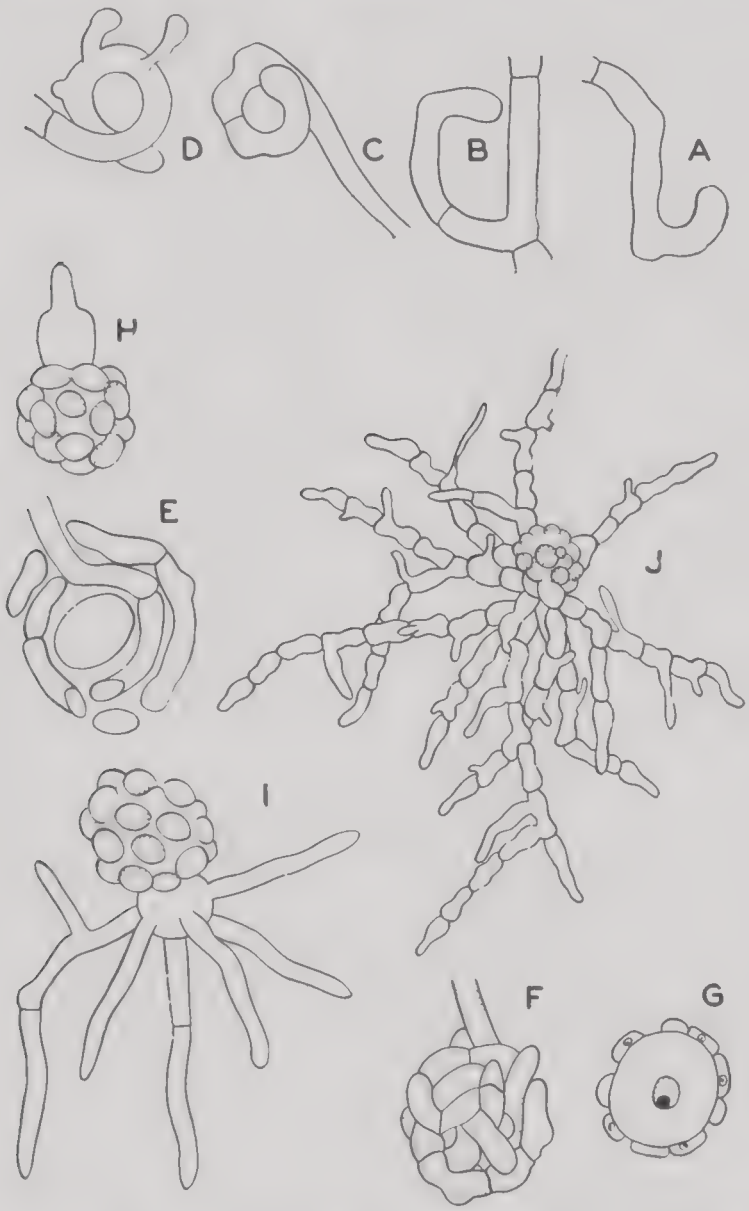

FIG. 119. *Urocystis cepulae*. *A–G*, successive stages in the development of the chlamydospore; *H J*, successive stages in germination of the chlamydospore and the formation of hyphal fragments. (*After Anderson.*)

described erroneously for *U. cepulae* by Thaxter (14), who apparently was confused by the germination of small spores of another fungus which had become lodged between the appendage cells. The first authentic germination of *U. cepulae* was described by Anderson (1). He found that the spores do not require a rest period, but they do not germinate well in distilled water, tap water, or soil water or in water to which onion tissue is added. On the other hand they germinate well on the surface of agar

plates, the oxygen relation appearing to be an important factor. The species is atypical of the genus since the spore upon germination gives rise to a short, hemispherical, hyaline basidium. No sporidia are formed, but mycelial branches grow out promptly from the basidium. As seen in pure culture, the branching mycelium continues to grow and become septate. Eventually the hyphal cells break apart into "hyphal fragments," which in turn germinate to produce new thalli and in a sense to function in place of sporidia (Fig. 119).

The nuclear history of the fungus was studied by Blizzard (4). The single nucleus of the mature chlamydospore divides in the spore during germination, and the nuclei migrate into the basidium, one passing eventually into each mycelial branch. The hyphal fragments are uninucleate, and the saprophytic mycelium remains uninucleate. The mycelium after infection becomes intercellular and remains uninucleate for a time but becomes progressively binucleate as the sporulation stage is approached. The compound spore originates with a group of binucleate sporogenous branches consisting of short cells. In the centrally located cell the nuclei fuse, and the cell enlarges rapidly, while the surrounding binucleate cells, serving as nurse cells, remain appressed to the surface of the central cell as sterile, probably dead, appendages. The fungus grows well on vegetable agars for an indefinite period although it has never been shown to produce chlamydospores in the saprophytic stage.

Disease Cycle. The fungus remains viable for an indefinite number of years in infested soil. Anderson (1) claimed that it persisted in the soil as a saprophyte. There is no convincing experimental evidence of this, however. Since the fungus is known to remain viable in air-dry soil for many years, it is possible that many of the spores remain dormant for a long time. Spores have been reported rarely on onion seed, and distribution by this means is not regarded as important. Onion sets, and to a lesser degree onion transplants, are important means of widespread distribution of the fungus.

The onion is susceptible to initial penetration in the seedling stage. Infection ordinarily occurs in the cotyledon before it emerges above ground (Fig. 120). When the cotyledon approaches full size, it is no longer susceptible. If by chance it has escaped infection during this early period, the entire plant continues to remain healthy. The leaves of the onion are produced successively in the embryonic transition region between root and leaf and remain enclosed within the preceding leaf until they emerge at the "neck" above ground. Each leaf goes through a susceptible period, but if the previous leaf has remained free from infection, the former is protected from infection until it passes into the resistant stage. If the cotyledon is removed before the first leaf has become resistant, direct

infection of the latter from the soil may occur, but ordinarily infection occurs first in the cotyledon, and the fungus penetrates each successive inner leaf. Often the infection of a cotyledon takes place only near the tip, or "knee," while it is below ground, but the lesion matures above ground. The base of the cotyledon then remains healthy and protects the first leaf from infection. Many originally infected plants escape systemic infection in this manner.

The optimum temperatures for chlamydospore germination, hyphal-fragment germination, and vegetative growth of the thallus lie between

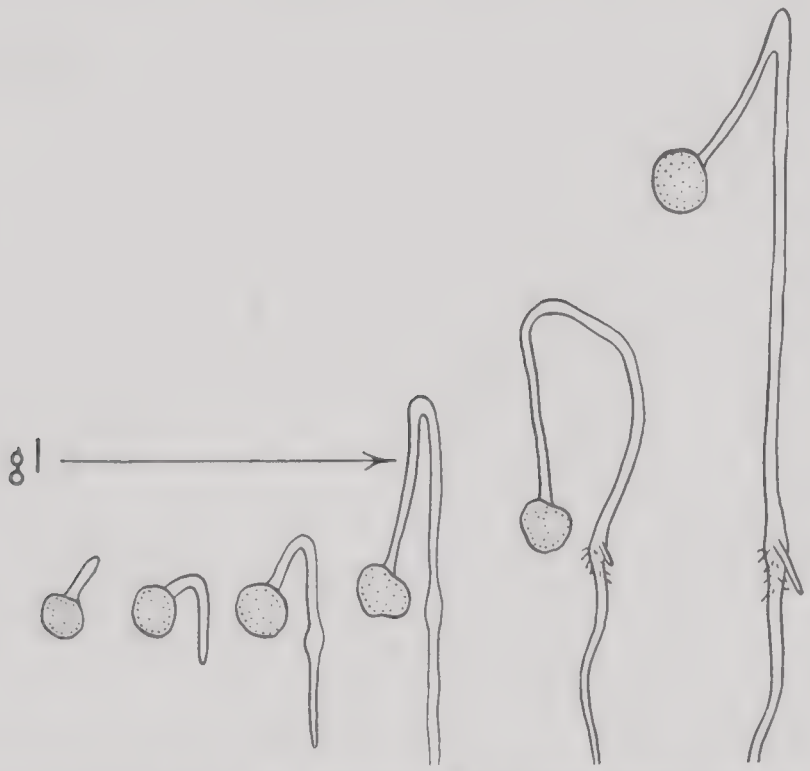

FIG. 120. Successive stages in the development of the cotyledon of an onion seedling; *gl* is the ground level. At the stage of the fourth plant from the left the cotyledon is approaching the immune stage. (*After Anderson.*)

13 and 22°C. Above 25° there is a decided reduction in the amount of germination which occurs, and growth of the thallus becomes more meager as the temperature rises. Such hyphae as do appear above 28° not only grow very slowly but lose their viability upon continued exposure to these temperatures. On the contrary, protracted exposure to near-minimum temperatures does not so affect the slowly growing thallus (18). Abundant infection occurs at soil temperatures as low as 10 to 12°, which is near the lower limit for germination and growth of the onion. Infection occurs equally well up to 25°, but above that there is a rapid reduction in infection, while at 29° the onion seedling grows free from the disease in infested soil. At soil temperatures slightly below 29° the seedling grows more

rapidly than at lower temperatures, and the susceptible period is correspondingly shortened. In the southern states onion seed is usually sown in autumn when the soil temperature is high. This is considered to be a major factor in restricting the disease in those areas (17).

Host Resistance. Evans has studied the host-parasite relations of this disease in onion (5) and in the relatively resistant Welsh onion (6). Penetration takes place directly through the cuticle, and dissolving action on the subcuticular wall follows (Fig. 121). The hypha enters the cell lumen, branches, and continues to penetrate surrounding cells. No haustoria are formed. As the mycelium progresses, it becomes intercellular, and the intracellular mycelium in the epidermal cells disintegrates, sometimes without permanent damage to the cell. The main development is then intercellular, the host cells being pushed apart as the lesion grows. The chlamydospores form in the intercellular mycelium, but the surrounding host cells have largely disintegrated by the time spores are mature.

When the fungus penetrates the onion cotyledon which is passing into the resistant stage, the mode of penetration is normal, but there are various signs of a change in host-parasite interaction. In many cases the mycelium rounds up into subcuticular vesicles without penetrating the epidermal cell. When the hyphae enter the epidermal or subepidermal cells, they often become contorted, misshapen, or irregularly swollen. The resistant character develops as a progressive incompatibility between the host and the intracellular and intercellular mycelium. In Welsh onion grown on heavily infested soil a high percentage of plants are healthy. Close examination shows, however, that a large percentage of plants become infected; in a majority of cases the lesions are near the tip of the cotyledon, and the plants escape systemic infection. Evans (6) suspected that this was due to a progression from the susceptible to the resistant stage earlier in Welsh onion than in common onion. Cytological study showed that the type of resistant reaction found in maturing cotyledons of common onion was present almost from the beginning in the tissues of the lower part of the cotyledon of the Welsh onion. Usually the only susceptible tissue was that near the tip, which situation resulted effectively in exclusion of the fungus from the embryonic leaves and provided a practical resistance to the pathogen.

No resistant individuals have been found in common onion, but the latter and Welsh onion cross fairly readily, although the hybrid is usually self-sterile. The hybrid may be backcrossed to each parent but least readily to common onion. Walker *et al.* (19) studied the inheritance of resistance in this hybrid and in a fertile amphidiploid from the hybrid. The F_1 hybrid was considerably more resistant than the susceptible parent but less so than the resistant parent. Backcrosses to Welsh onion were

highly resistant, but backcrosses to common onion were very susceptible except for an occasional survivor which might have further breeding value. The amphidiploid, which is a nonbulbing type like Welsh onion, is quite

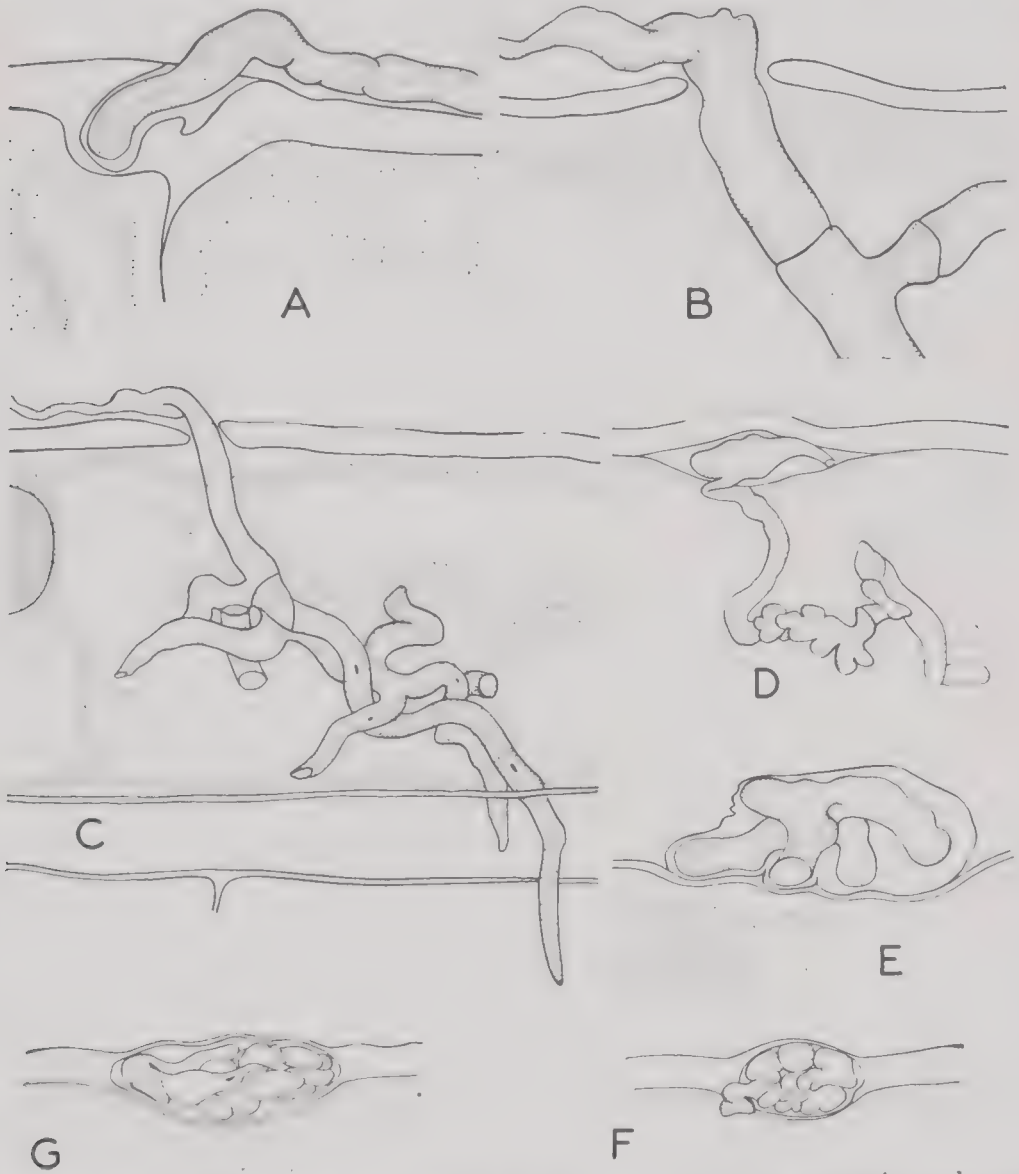

FIG. 121. *Urocystis cepulae*. *A*, early stage of penetration showing the result of apparent dissolving action of the hyphal tip upon material of the subcuticular layer of the epidermal cell wall; *B*, a later stage than *A*, the disintegrated portion of the infection hypha remaining outside the host; *C*, intracellular mycelium in the epidermal cell of susceptible tissue, older parts being dead and the advancing portion becoming intercellular; *D*, penetration at the time the tissue is beginning to become immune; a subcuticular vesicle formed, and an extension into the cell lumen has become devoid of cell contents; *E*–*G*, penetration after the tissue has become immune, resulting in the fungus becoming confined entirely or largely in the form of a vesicle beneath the cuticle. (*After Evans.*)

highly resistant, though less so than the Welsh onion. It has value as a bunching onion and is known under the name of Beltsville Bunching.

Control. Thaxter (14) was the first to show the short susceptible period of the seedling onion. He also was the first to try various materials

placed in the furrow with the seed as protectants. A mixture of sulfur and lime showed promise, and this treatment was placed on a commercial basis in New York by Sirrine and Stewart (13) in 1900. Shortly after this Selby developed the formaldehyde-drip control in Ohio (11, 12), which has been improved and applied widely since that time (15, 16). The latter method consists in applying a stream of dilute formaldehyde in the soil with the seed. Apparently the material volatilizes sufficiently to inhibit the fungus for the period of susceptibility in the soil through which the seedling emerges. The standard treatments consist of 1 pint of 37 to 40 per cent formaldehyde solution in 16 gal. of water applied at the rate of 1 gal. to 150 ft. of row or 1 pint in 8 gal. of water at the rate of 300 ft. of row (16). Limitations of this treatment are that it is cumbersome, that formaldehyde injury may occur in very dry soil (2), and that heavy rains immediately after application may reduce its effectiveness (8). Arasan was shown by Newhall in New York State to be effective when pelleted on the seed with methocel sticker (Chap. 16). In Wisconsin on upland soil good control was secured with 1 lb. of Arasan to 10 lb. of seed applied without sticker and with 1 lb. of the material to 4 lb. of seed applied with sticker (7). Nelson (10) secured erratic results with this method on muck in Michigan.

Onion sets and onion transplants may be planted in smut-infested soil without any danger of infection.

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THE PLANT RUSTS

The rust fungi comprise a single order (Uredinales) every member of which is an obligate parasite. From the standpoint of plant pathology the rusts are extremely important. In the small grains particularly, they comprise a group of major diseases. Two books by J. C. Arthur (1, 2), who was America's outstanding student of this order, are indispensible to the student concerned with this group.

Macrocyclic and Microcyclic Rusts. Two aspects of the life history of rust fungi should be understood at the outset. Most species have several spore forms, which appear in a definite succession. When a rust fungus has more forms than the **teliospore** and **sporidium,** it is referred to as a **long-cycle,** or **macrocyclic,** rust (*e.g.*, *Puccinia graminis* Pers.). The teliospore is common to all rust fungi, except those of imperfect-form species, and upon germination it gives rise to the **promycelium** (**basidium**). There follow, in order, **sporidia** (**basidiospores**), **pycniospores** (**spermatia**), **aeciospores, urediospores,** and finally **teliospores.** The spores other than sporidia are produced in sori, which are designated as **pycnia** (**spermagonia**), **aecia, uredia,** and **telia,** respectively. In all macrocyclic species aecia and telia are formed, but in some species pycnia and uredia are absent. Some rust fungi are known as **short-cycle,** or **microcyclic,** rusts. The microcyclic species may or may not have pycnia, and they have only one other type of sorus known as the **micro-telium** (*e.g.*, *Puccinia malvacearum* Bert.). It starts out like an aecium and in appearance ends up either like an aecium or as a telium. The spores germinate like teliospores and give rise to sporidia.

Heteroecious and Autoecious Rusts. Within the macrocyclic rusts two distinct types occur with relation to the host plant. Some species require two distinct host plants for the completion of the full life cycle. For instance, the black-stem-rust fungus (*Puccinia graminis*) produces

uredia and telia only on grains and grasses and pycnia and aecia only on barberry and mahonia. Such species are known as **heteroecious.** Other species in the same genus may produce all sori on the same host. As an example, *Puccinia asparagi* DC. produces all four types of sori on asparagus. Such species are referred to as **autoecious.**

While it is possible to treat only five rust diseases in this book, they are chosen as representative types. The two families in the Uredinales which comprise all rust fungi except the imperfect forms are given on page 371. The important rust diseases on cereals and grasses are treated by Dickson (3), and the literature on physiologic specialization in the cereal rusts was reviewed in 1946 by Johnson and Newton (4).

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BLACK STEM RUST OF SMALL GRAINS AND GRASSES

Black stem rust is one of the many rusts which affect members of the grass family. It has a wide host range including wheat, oats, barley, rye, timothy, and numerous wild and cultivated grasses. It is most important economically on wheat, on which crop it has been a subject of observation and study for centuries. The fungus is heteroecious, alternating from grass or cereal to barberry or mahonia.

While the fungus is almost coexistent with wheat throughout the world, the disease is most damaging in moderately moist areas and in moist seasons in areas with low average rainfall. In the United States and Canada the losses from black stem rust are highest in the spring-wheat area of the upper Mississippi valley and that portion of the Hudson Bay watershed immediately to the north. The nature and extent of losses incurred as a result of black stem rust are discussed by Craigie (11, 12) and by Greaney (17).

Symptoms. The uredial stage, often referred to as the red-rust stage or as the summer stage, appears on leaves and culms at any stage of development of grass or cereal (Fig. 122). It may occur also upon various parts of the spike and kernel. The uredia are usually elongate pustules exposing a reddish-brown spore mass, which in forming ruptures the epidermis of the host. The size, shape, and number of sori vary with environment and particularly with the inherent resistance of the host concerned. Various amounts of chlorosis surround the sori, and in very resistant hosts

no lesions or only necrotic flecks develop at the point of penetration. Teliospores appear in old uredia, replacing the urediospores, or they develop in independent sori as the host plants approach maturation. Telia are dark brown to black and are smooth rather than powdery, although the epidermis of the host plant is broken and the spores are exposed.

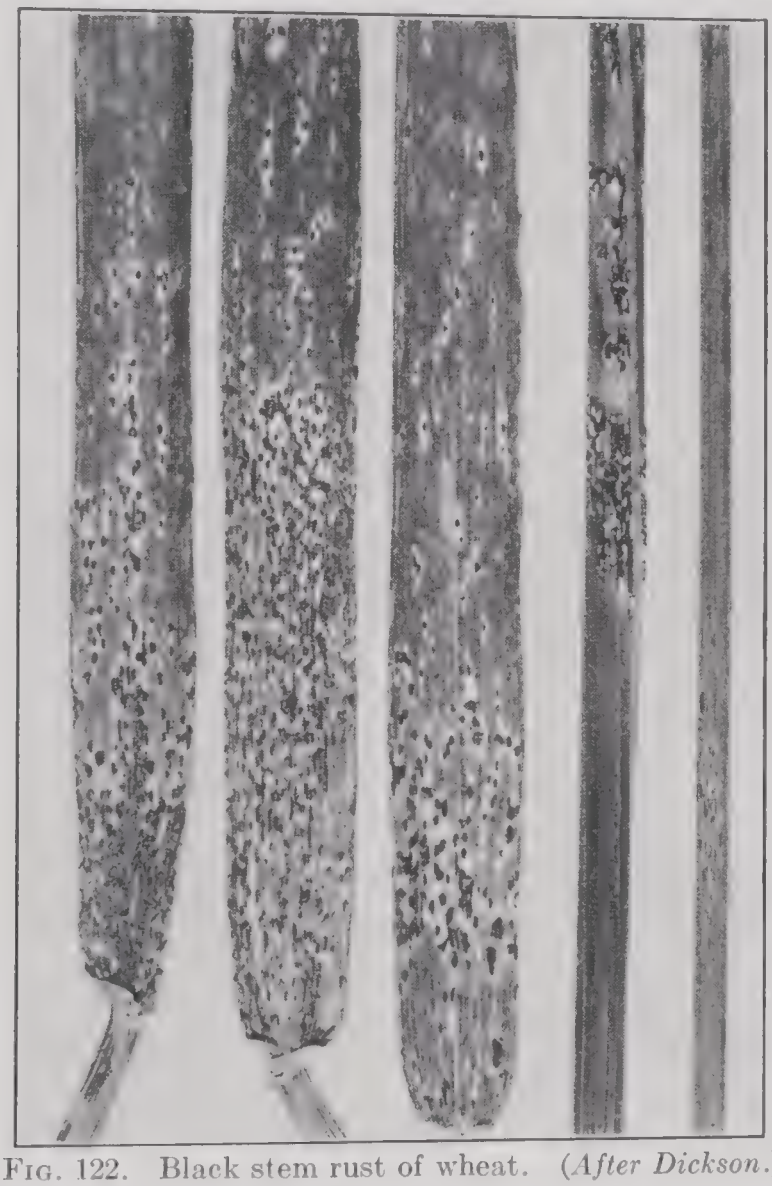

FIG. 122. Black stem rust of wheat. (*After Dickson.*)

The aecia form as groups of aecial cups on leaves, petioles, and flower parts of barberry or mahonia, the host tissue turning yellowish and often becoming hypertrophied (Fig. 123). The aecia are orange yellow, and the numerous tubular aecial cups have marginal light-colored peridia, which are contrasted with the orange-colored spore masses within the cups. Previous to the appearance of aecia there forms on the opposite side of the leaf an orange-yellow slightly elevated spot from which a sticky liquid exudes. This is the lesion in which the pycnium is located, the ostiole protruding to the surface and liberating the exudate.

The Causal Organism. *Puccinia graminis* Pers., 1794.

SYNONYMY:
Lycoperdon poculiforme Jacq., 1786.
Lycoperdon lineare Schrank, 1789.
Aecidium berberidis Pers., 1791.
Uredo linearis Pers., 1801.
Puccinia linearis Röhling, 1813.
Puccinia poculiformis Wettst., 1886.
Dicaeoma poculiforme Kuntze, 1898.

On the cereal or grass host the uredia are oblong to circular, orange red to chestnut brown, and powdery (Fig. 124). Urediospores (13 to 24 by 21 to 42 μ) are oblong or ellipsoid with four equatorially arranged germ pores, golden brown, and strongly echinulate. Telia form in uredia or independently as the host approaches maturity. They are oblong to linear, dark brown to black, and not powdery although the spores are exposed. Teliospores are ellipsoid or oblong-clavate (16 to 23 by 35 to 58 μ), two-celled, slightly constricted at the septum, rounded to slightly pointed with a thickened wall at the apex, smooth, and dark chestnut brown. They are borne on persistent pedicels and germinate *in situ* after a rest period to form a hyaline four-celled promycelium (basidium), on which are borne hyaline, continuous, sporidia (basidiospores) on sterigmata (Fig. 125).

FIG. 123. Aecial cups of black stem rust on barberry. (*After Dickson.*)

The pycnia are flask-shaped, appearing embedded in the tissue in orange-yellow spots on the upper sides of leaves of barberry or mahonia (Fig. 124). They contain paraphyses and hyaline, slender, continuous pycniospores (spermatia), which emerge in a viscous exudate through an ostiole along with flexuous hyphae. The aecia appear on the lower surfaces of leaves of barberry and mahonia after the full development of the pycnia on the upper side. The lesion is elevated and yellowish, while the aecia are orange yellow and consist of numerous tubular aecial cups containing orange-yellow powdery masses of aecio-

spores, each cup being surrounded by a light-colored peridium. The latter varies in length with the environment and at the tip is turned outward with a serrated margin. The aeciospores, borne in chains within the cup, are angular, globoid, or oblong (15 to 19 by 16 to 23 μ), light orange yellow, and smooth.

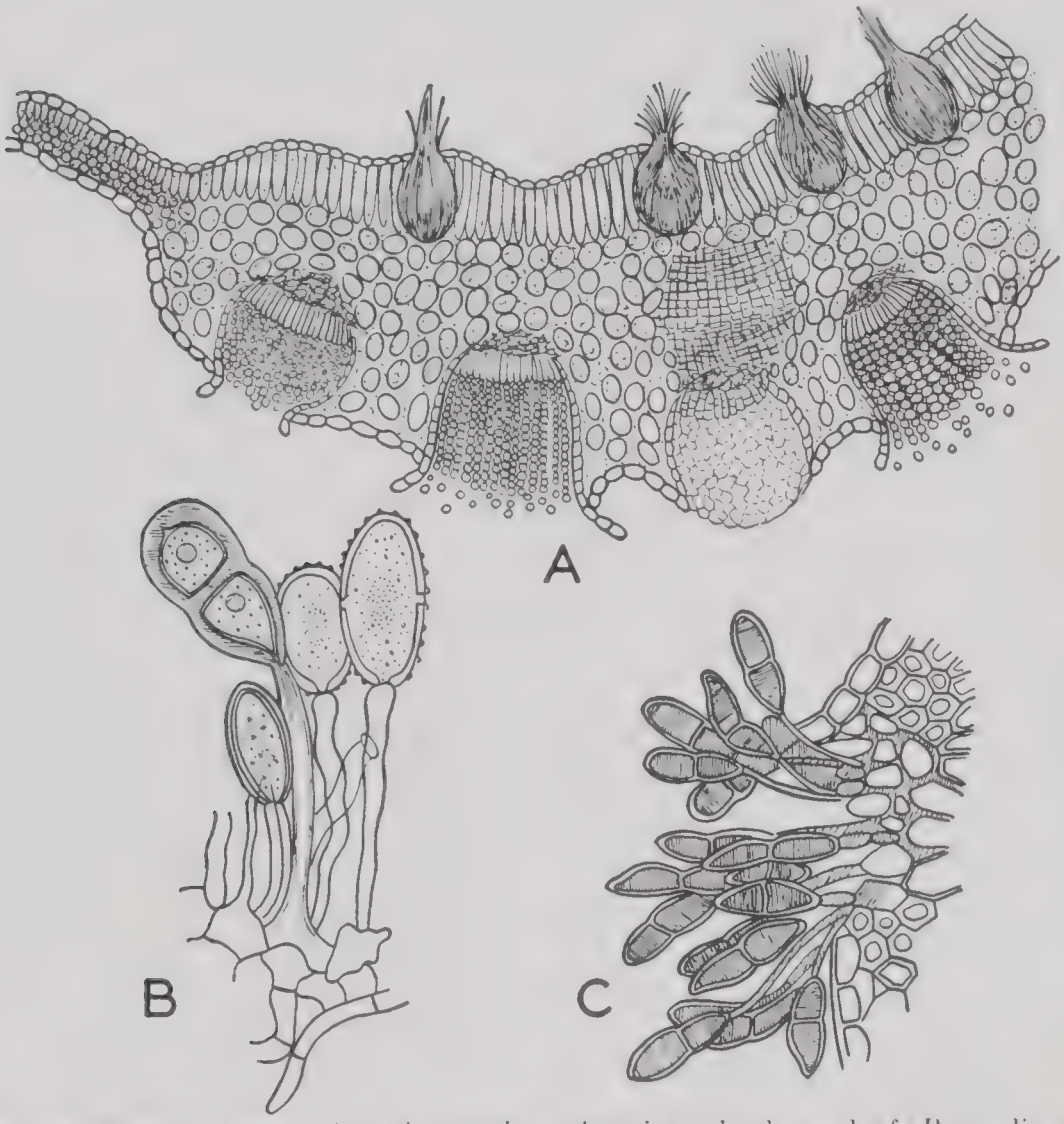

FIG. 124. *Puccinia graminis*. *A*, pycnia and aecia on barberry leaf; *B*, urediospores and a teliospore arising from the same sorus; *C*, teliospores arising from a telium. (*After Sachs.*)

In 1794 Persoon, in an attempt to set up an arrangement of fungi, recognized three genera of the rusts, *i.e.*, *Puccinia*, *Uredo*, and *Aecidium*. In *Puccinia* he placed telial forms of many species, some of which are now classed in other genera, and he included the telial forms of the black stem rust under the name *Puccinia graminis*. The fungus had been previously named *Lycoperdon poculiforme* by Jacquin in 1786 and *Lycoperdon lineare* by Schrank in 1789. The form on barberry was named *Aecidium berberidis*

by Persoon in 1791. In 1801 Persoon recognized the uredial stage as the species *Uredo linearis* and the telial stage as a distinct species, *Puccinia graminis*. Meyen later suggested that the pycnia were male organs. However, the function of the pycnia was not established until the work of Craigie (10) in 1927. The Tulasnes (50) in 1847 and De Bary (13) in

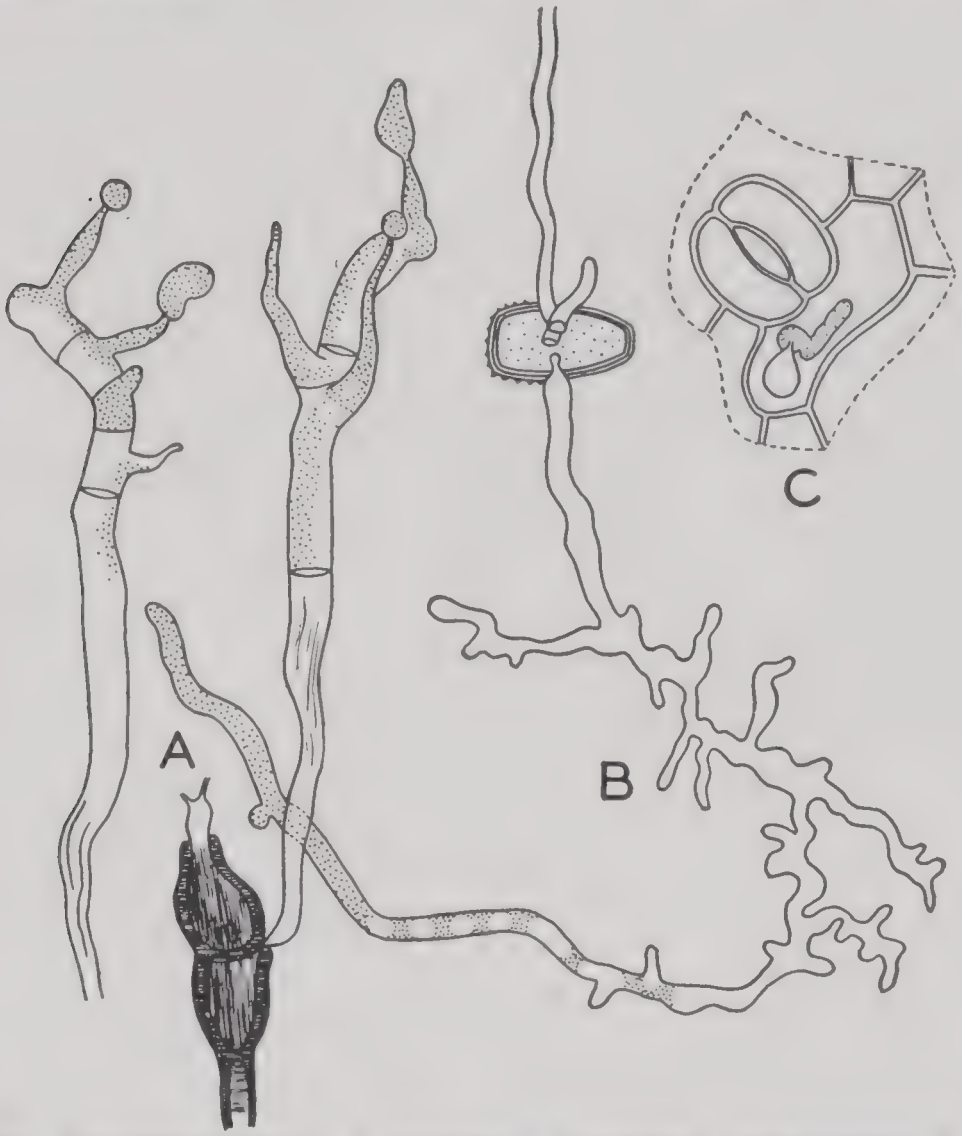

FIG. 125. *Puccinia graminis*. *A*, teliospore, promycelia, and sporidia; *B*, germinating urediospore; *C*, direct penetration of barberry leaf by germ tube of a sporidium. (*After Sachs.*)

1853 did not recognize that the uredial and telial stages were parts of a polymorphic species. The relation of the two forms was established by Tulasne (49) in 1854. In 1853 De Bary (13) strongly opposed the belief that the forms on wheat and those on barberry were connected in any way. However, in 1865 he provided the first demonstration of heteroecism in the rusts when he showed that the forms of *P. graminis* on cereals and of *Aecidium berberidis* on barberry were parts of the life cycle of the same organism (14).

The teliospore is the only truly diploid stage, becoming so as the two haploid nuclei in each cell fuse during the maturation of the spore. Upon germination by the production of the promycelium the nuclei become haploid by the reduction division (meiosis), and haploid nuclei move into the sporidia. The latter are borne by air currents, and when they contact the alternate host (barberry or mahonia), they germinate and penetrate directly (53). The mycelium forms the pycnium, and in due time the pycniospores and flexuous hyphae exude from or grow out of the ostiole

FIG. 126. *Puccinia graminis*. *A*, newly opened pycnium showing uninucleate paraphyses and pycniospores; *B*, formation of uninucleate pycniospores from the sporophore; *C*, binucleate paraphyses after fertilization. (*After Allen.*)

(Fig. 126). Hyphae may also reach the surface of young leaves through stomata or between epidermal cell walls (4, 5). In the course of meiosis in the promycelium the haploid lines segregate into two intercompatible but nonintracompatible mating groups (+) and (−). This is the discovery of Craigie in 1927 which established heterothallism in this species (10). Mating between the structures of (+) and (−) thalli is necessary, therefore, before the life cycle of the fungus proceeds beyond the pycnial stage. Each pycnium and its pycniospores and flexuous hyphae are either (+) or (−). The pycniospores in the viscous exudate are transmitted to other exudate drops by insects. When a (+) pycniospore comes in contact with the flexuous hypha of a (−) pycnium, fusion occurs, and the (+) nucleus migrates into the hypha (8). Multinucleate and binucleate

cells appear in the mycelium in the tissue, and the binucleate cells of the aecial primordium (protoaecium) each contain a (+) and a (−) nucleus (4, 5). Development of the aecium is not completed until the mycelium becomes binucleate. The aeciospores which form are binucleate, but the nuclei are still haploid. The thallus containing the two types of nuclei is referred to as dicaryotic, in contrast with the monocaryotic (uninucleate) mycelium. The mycelium from the aeciospore will not infect barberry but will infect the cereal or grass host. On this host it remains dicaryotic,

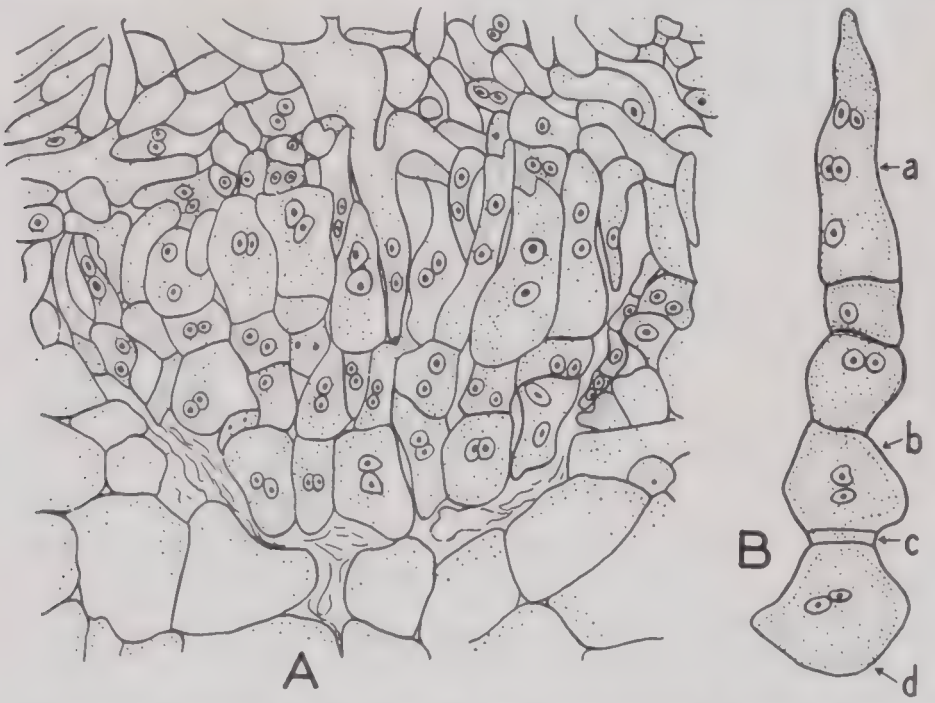

FIG. 127. *Puccinia graminis*. *A*, fertile aecium showing young binucleate spore chains before the cup has opened; *B*, spore chain with multinucleate basal cell (*a*), spore mother cell (*b*), intercalary cell (*c*), and aeciospore (*d*). (*After Allen.*)

giving rise to the uredia and telia. The true diploid stage is reached when the (+) and (−) nuclei fuse in the maturing teliospore.

The fungus was shown by Eriksson and Henning (15, 16) to consist of subspecies which differ little in morphology but are specialized in the dicaryotic stage to certain groups of graminicolous hosts. These authors set up subspecies which are designated by trinomials and are specialized in their pathogenic relations to certain economic cereals and grasses. The currently used trinominals follow:

Puccinia graminis tritici Erikss. & E. Henn.
Puccinia graminis secalis Erikss. & E. Henn.
Puccinia graminis avenae Erikss. & E. Henn.
Puccinia graminis agrostis Erikss.
Puccinia graminis phleipratensis (Erikss. & E. Henn.) Stakman & Piemeisel.
Puccinia graminis bromi Erikss.
Puccinia graminis poae Erikss. & E. Henn.
Puccinia graminis airae Erikss. & E. Henn.

Slight but relatively consistent differences between the subspecies in size of the various spore forms were determined by Levine (34). He also noted that the spores tended to be smaller when produced on resistant hosts and when produced under adverse environmental conditions. As shown below, there exist numerous physiologic races within each subspecies. Morphological differences were found by Levine (35) sometimes to be as great between races within *tritici* as between *tritici* as a whole and other subspecies. As a matter of practice, however, the identification of races and subspecies has been found to be based most satisfactorily upon their parasitic effect upon selected varieties of the host plant concerned.

Disease Cycle. In the northern part of the United States and in Canada and in areas elsewhere comparable as to climate, the organism overwinters as teliospores on wild grasses and in grain stubble and straw. These germinate *in situ*, producing sporidia, which are carried by air currents to barberry, where young stems, petioles, and leaves are infected following direct penetration of the cuticle (53). The mycelium, at first intracellular, becomes primarily intercellular and promptly forms the pycnia chiefly beneath the upper epidermis of the leaf (4). Pycniospores are formed within the pycnia and are exuded through an ostiole to the leaf surface in a viscous liquid. Flexuous hyphae grow through the ostiole, and some mycelial hyphae reach the surface through stomata. Aecial primordia begin to form, but their maturation does not proceed until pycniospores from a compatible type are transferred to the surface of the lesion by insects or by spattering rain. Following fusion of introduced pycniospores with flexuous hyphae, the initiation of the dicaryotic stage results in formation of aeciospores. The latter are carried by air currents to the graminicolous host. They germinate, form appressoria, penetrate always through the stomata, and each forms a vesicle in the substomatal chamber from which hyphae proceed intercellularly into the parenchymatous tissue. As a hypha contacts the outer wall of the host cell, it swells at the tip; the two nuclei divide; a septum marks off a short terminal cell (the haustorium mother cell) which is closely appressed to the host-cell wall. A fine pore forms through the walls of fungus and host; the contents of the fungus pass into the host cell, forming the haustorium (1). In the congenial host the mycelium within a few days gives rise to the uredia and urediospores. The latter are air-borne and can infect only the graminicolous host in the same manner as the aeciospore. The urediospore cycle may be repeated several times during the season, comprising the summer red-rust stage, which causes the chief damage. In uredia and in other spots, the mycelium gives rise to telia and teliospores as the host plant approaches maturity.

One of the first recorded attempts to demonstrate a connection between

the common barberry and wheat rust was made by Thomas Andrew Knight, English horticulturist and plant breeder. He sowed wheat around a barberry bush in the autumn of 1804, and some of the same lot of seed at a distance. In the following July rust appeared on the wheat near the barberry but not on those plants at a distance until Knight carried a branch of the rusted barberry to them. He sprinkled water over the barberry branch in proximity to the wheat on three successive days and recorded rust on the wheat 10 days later. Shøler in Denmark in 1813 to 1817 planted barberries at the edge of grain fields and showed that rust was most severe near the bushes. He repeated Knight's experiment by taking branches of rusted barberry plants and brushing them on rye plants wet with dew. He noted rust lesions on marked plants after 5 days. While cultivators and laymen for generations had believed in the association of the diseases of the two widely different plants and had from time to time passed laws designed toward eradication of the barberry both in Europe and in America (Chap. 15), mycologists held aloof from such an interpretation until the experimental evidence of heteroecism was presented by De Bary in 1865 (14).

As one proceeds into the middle belt of states and farther to the south, the percentage of overwintering urediospores increases, particularly on winter-grown grain. In some southern regions the most unfavorable periods for survival of urediospores may be during hot grain-free periods in the summer. In northern states the teliospores function in continuation of the life cycle of the organism only when barberries are available at the time of their germination. Barberries are most susceptible when leaf and stem tissues are young (37). As one proceeds south, the importance of the barberry decreases, largely because it does not expose susceptible tissue at the time sporidia are available and because teliospores produced in the southern, relatively warm winter areas do not germinate.

The eradication of the barberry in the North serves to break the cycle at the time of teliospore germination since the sporidia can not infect graminicolous hosts. In the South, where urediospores are formed on the winter grain in early spring, the latter spores are carried northward by air currents. It is possible, therefore, for the fungus to migrate northward with the crop, and epidemics in northern states are nowadays induced largely by inoculum from that source (47). The exact timing of favorable environmental conditions for reproduction of the uredo cycle and for dissemination of spores is required. Usually the migration is prevented or retarded by some periods of unfavorable environment.

Levine (35) made a study over a 5-year period (1919–1923) of the occurrence of the disease on wheat in the upper Mississippi valley. He found a positive correlation between mean temperature and total rainfall

during the last 2 months of the growing season and severity of black stem rust on susceptible varieties. When the total precipitation fell below 2 in. or when the mean temperature was less than 60°F., no rust developed, while severe stem rust was observed only when the mean temperature ranged between 66° and 72° and the total rainfall exceeded 2.5 in. In other words warm moist weather is necessary for the production of abundant inoculum when susceptible hosts are available. The same general relation of weather to epidemiology was noted by Craigie (12) in Canada. Johnson and Newton (27) showed that when infected plants were grown in the greenhouse at temperatures above the optimum, they tended to show various degrees of resistance, as evidenced by smaller pustules.

The mycoplasm theory of Eriksson is now a matter of historical interest. It was put forth in the last decade of the nineteenth century to explain a means of overwintering of the fungus. The theory was based on a concept that in fall-sown grain, as an example, the fungus cytoplasm in the haustorium merged with that of the host cell in a manner which rendered them indistinguishable. Although the fungus had "disappeared," it continued to exist. At a later time the fungus protoplast again took form and proceeded pathogenetically to invade the host tissue and produce uredia in a normal manner. The mycoplasm theory was refuted by Ward (52) and has now been generally discarded.

Physiologic Races. Eriksson and Henning (15, 16) defined subspecies of *Puccinia graminis*, as indicated above, in 1894 and 1896. In a study of brome rust, *P. rubigo-vera* (DC.) Wint., in the same decade, Ward (52) in England showed a similar type of specialization toward species of *Bromus*. There were, however, distinct indications of intermediate hosts susceptible in various degrees to more than one race. Ward believed that the host substrate influenced the pathogenicity of a given race. He believed that continued culture of a race on an uncongenial intermediate host tended to change the race to the point where it would infect a previously immune host. This concept became known as Ward's "bridging-host" theory. Stakman (42) studied this question thoroughly with reference to *P. graminis* and concluded that there was no evidence of bridging hosts with this species. In fact, Ward's theory has by now been discarded as generally untenable.

In 1917 Stakman and Piemeisel (43) noted that collections of rust on club wheat from Idaho and Washington had a different infective capacity on varieties of bread wheat than that commonly encountered with collections from the Mississippi valley. Melchers and Parker (38) in Kansas reported during the next year another race of *P. graminis tritici*. These investigations were the beginning of an intensive study of physiologic races in this and many other species of rusts. Stakman and associates at

the University of Minnesota have led in development of methods for differentiation of races of *P. graminis*, especially *P. graminis tritici*. Recognizing that races were best defined by the effect upon different hosts, Stakman and Levine (43) laid out the bases in 1922. Twelve wheat varieties from five species of *Triticum* are used as differential hosts. These are as follows:

Species	Variety	Accession No.
T. compactum Host	Little Club	C. I. 4066
T. vulgare Vill.	Marquis	C. I. 3641
T. vulgare	Reliance	C. I. 7370
T. vulgare	Kota	C. I. 5878
T. durum Desf.	Arnautka	C. I. 1493
T. durum	Mindum	C. I. 5296
T. durum	Spelmer	C. I. 6236
T. durum	Kubanka	C. I. 2094
T. durum	Acme	C. I. 5284
T. monococcum L.	Einkorn	C. I. 2433
T. dicoccum Schrank	Vernal	C. I. 3686
T. dicoccum	Khapli	C. I. 4013

The types of infection and their symbols are as follows:

0 *Immune.* No uredia; hypersensitive flecks when present indicated by a semicolon.

1 *Very resistant.* Uredia minute, scattered, surrounded by necrotic areas.

2 *Moderately resistant.* Uredia small to medium in size, isolated; pustules usually in green islands surrounded by a decidedly chlorotic or necrotic border.

3 *Moderately susceptible.* Uredia medium in size; coalescence infrequent; no necrotic areas; chlorotic zones may be present, especially under unfavorable growing conditions.

4 *Very susceptible.* Uredia large, numerous, confluent; no necrosis; chlorosis may be present under unfavorable cultural conditions.

X *Heterogeneous.* All types of infection present on same leaf blade.

The present methods are described by Stakman *et al.* (48). About 200 distinct races within *P. graminis tritici* are now known. The same pattern for differentiation of races within species has been followed in most of the cereal rusts and in certain others.

In general, races of *P. graminis tritici* described in this manner are quite stable, although occasional mutations are observed (40). It is now recognized that more than one biotype may be contained within a race. The basis for this has been determined by study of race hybrids. When heterothallism was discovered, a means for crossing races was provided. Johnson and Newton (24, 26, 28, 29, 31, 33, 39, 40) led in this field. They

found that it was possible to cross subspecies of *P. graminis*, but this was not usually so successful as hybridization of races within a subspecies. Such characters as spore color were found to be controlled by gene pairs, and the same was true for pathogenicity. We may now well refer to the review by Buller cited early in this chapter; in this discussion it was pointed out that in many Basidiomycetes it has been shown that a dominant gene in one genom of the dicaryotic mycelium expressed itself over the recessive member of the pair in the second genom even though the genoms had not combined in a $2n$ nucleus. In the rust mycelium in wheat, then, if a given type of infection on a given test variety were controlled by a dominant gene, the property would be expressed if this gene were present in one or both genoms. Thus different collections of a given race (*e.g.*, race 15) might be similar phenotypically but different genotypically. This is exactly what Johnson and Newton found. If two collections corresponded to the reaction of race 15 on the 12 differential hosts, they might be somewhat different on a thirteenth variety, or they might react differently in environment outside the ordinary range. This also has been found to be the case. Some collections of race 15 are very virulent on some old and some new varieties which are highly resistant to other (typical) collections of race 15. It has become necessary, therefore, to set up classes within some races, and in the case of race 15, the common type is known as 15A and the more virulent one 15B (20, 21, 25). In all probability 15A and 15B are distinct genotypically.

Varietal Resistance. From what has been stated in the previous paragraphs on physiologic variation it is obvious that the large number of races greatly complicates the problem of developing varieties resistant to black stem rust. An immense amount of research in this field has been carried out, and much is still under way. Many studies with selected varieties and races have shown that high resistance to any one race is usually controlled by a dominant gene in a gene pair. There seems to be no barrier to eventually bringing together genes for resistance to all races within one or more varieties. However, the practical aspects of breeding programs make it essential to focus attention on the most prevalent and important races in a given region and to combine resistance to them with resistance to other rusts, to smuts, and to other diseases. The progress in breeding for resistance in the cereal crops has been summarized up to about 1942 by Ausemus (6).

It is to be expected that breeding for rust resistance, as in other cases of disease resistance, is a continuing process, because new races may appear as a result of mutation or recombination and because a foreign race, not new, may become established in the region concerned. In the upper Mississippi valley, for instance, a number of varieties have come forward and receded

largely because of appearance of races virulent upon them or because of extreme susceptibility to other diseases (7).

When a resistant variety is exposed to urediospores or aeciospores, germination and formation of appressoria proceed in the same manner as on the susceptible host. More stomata may be entered in the very susceptible than in the very resistant plant. In the congenial host the mycelium arising from the substomatal vesicle proceeds intercellularly, forms haustoria, and gradually retards growth and productivity of the host. In the resistant host the same process occurs up to the formation of a small haustorium. Then the latter collapses, as does the haustorium mother cell, and in turn the host cell dies. In the highly resistant host, then, little damage is done (1–3). The type of resistance just described is based upon the interaction of the protoplasts of host and pathogen. It is commonly referred to as protoplasmic resistance and is a type which is commonly found in many rust diseases. Another type which occasionally influences stem-rust resistance in wheat has to do with opening and closing of stomata. Some rust fungi will enter their hosts about as well in the dark as in the light. This is not usually the case with black stem rust of wheat, where daylight hours are most favorable for infection (22, 23). Humidity is highest, however, in the early morning hours as stomata are opening. The critical time, then, is that period when dew is still present and stomata are open. Stomata open more tardily in some varieties than in others. Hart (18) showed that in the resistant variety Hope, delayed opening of stomata was one factor contributing to its resistance. This type of resistance has been referred to as functional resistance. It is of relatively minor importance compared with protoplasmic resistance.

A third type of resistance to stem rust in wheat is known as morphologic resistance, or mature-plant resistance. In 1890 Cobb (9), studying resistance of wheat to stem rust in Australia, pointed out some correlations between the anatomical structure of wheat and resistance. Some 35 years later, after races became well defined, it was noted by breeders that lines susceptible to a race in the seedling stage sometimes showed marked resistance in the field where the plants were not exposed to that race until they were approaching maturity. It gradually became common knowledge that mature-plant resistance and seedling resistance were inherited independently. The nature of mature-plant resistance was studied critically by Hart (19), who showed that it was bound up with the relative amount of thick-walled collenchyma which developed in a given host line as it approached maturity. The rust fungus does not generally invade collenchyma, and when a minimum of parenchyma is available, the damage the fungus can cause at this most critical stage is reduced accordingly. A given variety may be susceptible to a given set of races in the seedling

stage but highly resistant to them in the mature-plant stage. For this reason, both protoplasmic resistance and mature-plant resistance are important to the breeder.

Environment may influence the expression of resistant characters, and in extreme cases a variety which ordinarily gives a 1 or a 2 reaction to a given race may give a 4 reaction. Reaction to different races may respond differently to environment (22, 23, 27, 30, 32, 36). Under especially favorable environment a variety resistant to a given race may show 4 reaction in the region just above the nodes, and by use of artificial techniques such regions may become susceptible to rusts not regarded as pathogens of the plant concerned under natural conditions (39). It is important, therefore, to bear in mind that resistance may be altered through the play of environment on host, pathogen, and their interaction.

All physiologic races, in so far as is known, infect barberry. When this host is infected, the pathogenic difference between races no longer holds. It is important to emphasize here that the monocaryotic mycelium of rusts has as a rule pathogenic properties distinct from those of the dicaryotic mycelium. In *Puccinia graminis* it not only requires a quite distinct host, but it invades the latter directly rather than through the stoma. In an autoecious rust, such as *Uromyces phaseoli typica*, Arth., the monocaryote penetrates directly, while the dicaryote penetrates through stomata (Fig. 183). Physiologic races in so far as barberry is concerned are unknown. Species of barberry differ, however, with regard to resistance. Generally, susceptible organs become resistant with age. Melander and Craigie (37) found that the increase in resistance with age is correlated with increase in resistance of the cuticle to puncture. Some species are immune, *e.g.*, the Japanese barberry (*Berberis thunbergii* DC.) and certain of its hybrids with the common species (*B. vulgaris* L.). In resistant species the cuticle is usually relatively resistant to puncture. This does not explain all cases of resistance, however, and in some cases the resistant property is associated with the protoplasm of the host.

Control. The two important approaches to control of black stem rust are through eradication of the barberry in northern regions and through the development of resistant varieties. The basis of and progress in eradication of the alternate host is discussed in Chap. 15. Eradication of barberries not only serves to remove current-season infection of grain by aeciospores, but also, in the long run even more important, it eliminates the possibility of recombination of genes in the rust and the production of races new to the locality concerned (12, 41, 45–47).

The basis for control through resistant varieties is discussed in the two previous sections. Numerous varieties resistant to important races in certain regions are available, and since breeding is a continuous process,

improvements and replacements will become available from time to time (6, 7).

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HOLLYHOCK RUST

This disease is of particular interest here because, in contrast to black stem rust, it is incited by a microcyclic species of *Puccinia*. It was first recorded in Chile in 1852. It was found in Australia in 1857, in several European countries between 1869 and 1874, and in North America in 1886. It is now world-wide in occurrence. Kellerman in Germany first recognized it as a short-cycle rust in 1874. A number of species of *Althea*, *Lavatera*, *Malva*, all genera of the Malvaceae, are reported as hosts.

Symptoms. Orange pustules appear on leaves, petioles, and stems. They are not powdery but are almost waxy on the surface and turn dark-colored with age. When sori are numerous and closely crowded, distortion of petioles may occur, and leaves may drop prematurely. Heavy infection results in unthrifty growth, but its chief damage is in the unsightliness of the ornamental host.

The Causal Organism. *Puccinia malvacearum* Bert., 1852.

Synonymy:
Puccinia malvacearum Mont., 1852.
Micropuccinia malvacearum Arth. & Jacks., 1921.

Pycnia do not occur. Telia are light chestnut brown. Teliospores (16 to 26 by 40 to 70 μ) are two-celled, oblong to fusiform-oblong, pedicellate. They germinate by production of a promycelium, followed by formation of sporidia. The teliospores form from binucleate basal cells, which may branch, each branch giving rise to a spore (Fig. 128). The spore initially divides into spore and stalk cell, and the spore cell divides again to form the two-celled spore. The nuclei fuse in each spore cell.

Upon germination the nucleus divides, and the promycelium divides first into two uninucleate cells and after another nuclear division into four uninucleate cells. A broad beak near the upper end of each cell swells at the tip to form a sporidium. The nucleus moves into the sporidium and divides as the spore is detached, the mature sporidium being binucleate (1). Ashworth (3) grew the fungus from single sporidia and found it to produce normal teliospores, showing that this species is homothallic.

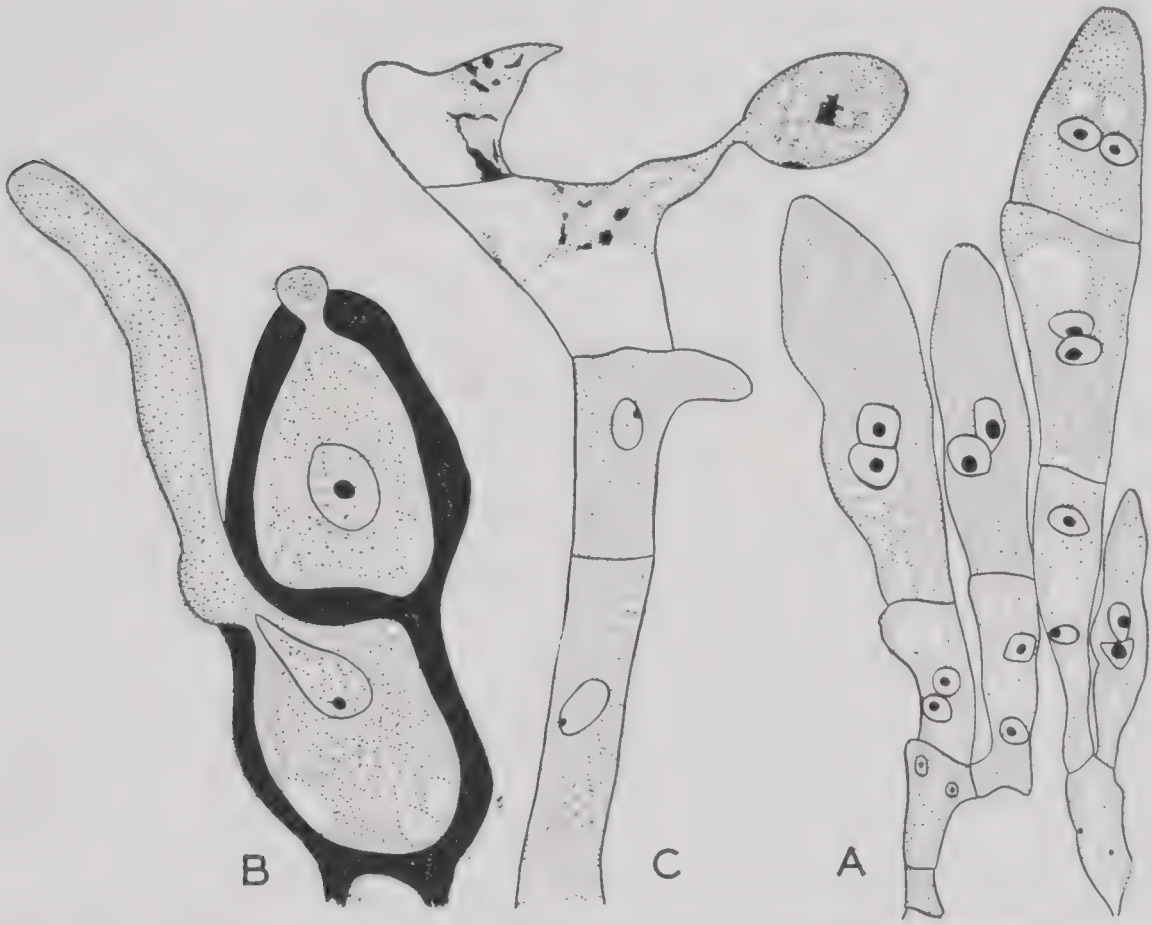

FIG. 128. *Puccinia malvacearum*. *A*, formation of binucleate teliospores from binucleate basal cells of the mycelium; *B*, mature teliospore, with diploid nucleus in each cell, in early stage of germination; *C*, formation of the uninucleate sporidium after meiotic division of the diploid nucleus in the promycelium. (*After Allen.*)

Allen (2) studied the nuclear history of the fungus more recently and found that whenever two monosporidial hyphae meet, anastamoses form, and an exchange of nuclei occurs. Thus it is possible for conjugation to occur.

Disease Cycle. Teliospores may germinate as soon as they are formed. Sporidia in contact with the epidermis of the host germinate, and the germ tube penetrates the cuticle and subcuticular wall (Fig. 129). Only rarely is a stoma entered. The cytoplasm flows from the germ tube into the mycelium within the host cell. A multicellular primary hypha forms and branches enter the palisade cells. The mycelium as it grows is partly

intercellular and partly intracellular. Although the sporidium is binucleate, the mycelium in the host becomes uninucleate and often remains so until the early stages of sorus development. Some hyphae grow out through the epidermal cells to the surface, where they may form conidium-like cells (2). No pycnia, aecia, or uredia are formed.

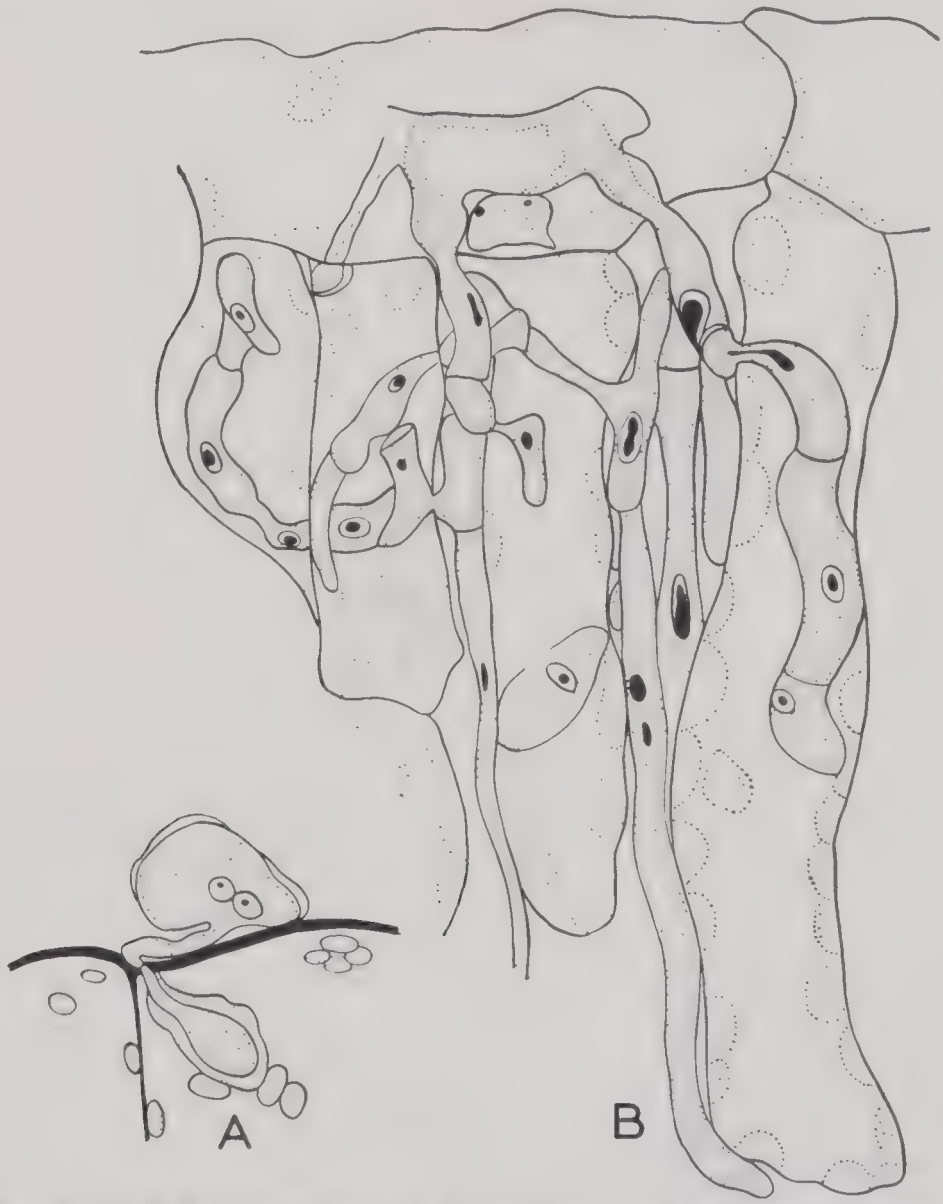

FIG. 129. *Puccinia malvacearum*. *A*, direct penetration by germ tube of a sporidium; *B*, intracellular uninucleate mycelium in the epidermal and palisade cells; the advancing hyphae have become intercellular. (*After Allen.*)

Taubenhaus (6) found the fungus to overwinter as mycelium on overwintering plants and as hibernating teliospores on old living or dead leaves and on the seeds or bracts. It is not uncommon to find the disease severe in the spring, declining in midsummer, and becoming severe again in autumn. Eriksson (5) attempted to explain this on the basis of two types of spores, autumn and summer spores. The latter are said to germi-

nate by long germ tubes which produce bud-spores, or conidia. When this latter spore germinates, it is said to fuse with the cytoplasm of the epidermal cell to form mycoplasm. These concepts have been refuted by Bailey (4).

Control. The chief means of control is to remove all infected plant material in the autumn in order to reduce the amount of overwintering inoculum. Dusting with sulfur during the growing season is helpful.

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RUST OF APPLE AND CEDAR

This rust is of importance on both of the alternate hosts, apple and red cedar. *Gymnosporangium juniperi-virginianae* Schw. is the most important

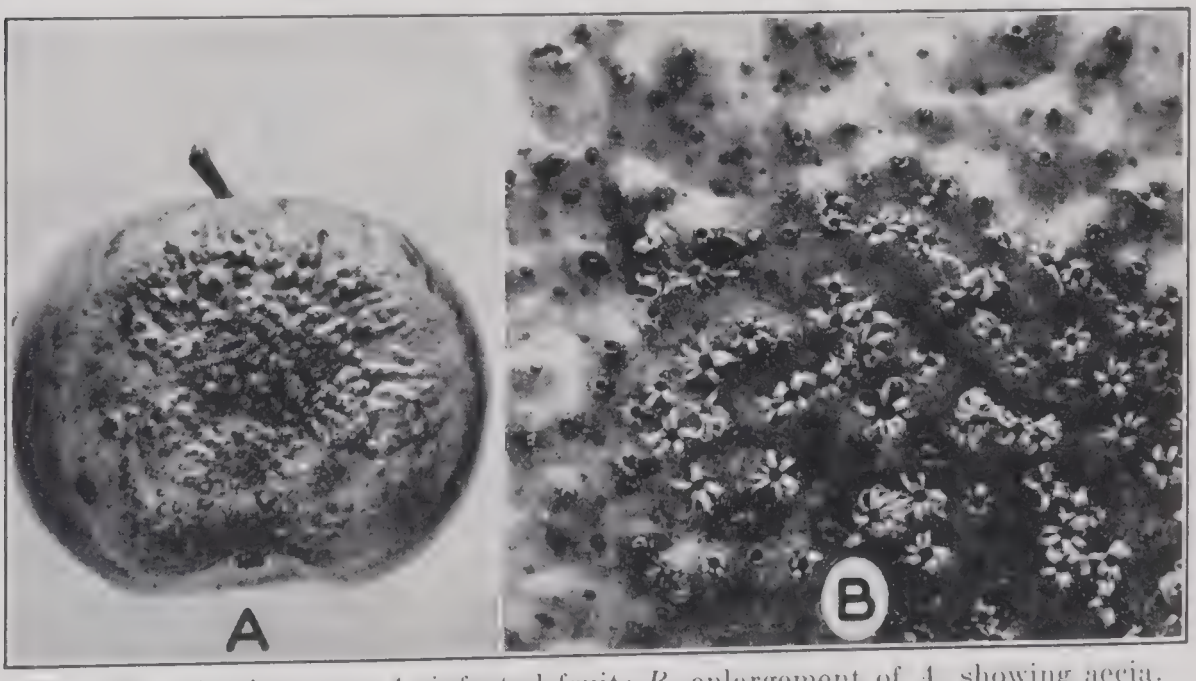

Fig. 130. Apple rust. *A*, infected fruit; *B*, enlargement of *A*, showing aecia.

of several species of the genus which infect apple in North America. While the rust is macrocyclic, it has only three of the possible four types of sori, *i.e.*, telia, aecia, and pycnia. The first occurs on the cedar; the last two

on apple. The disease is most important on apple when orchards are located where red cedar (*Juniperus virginiana* L.) is native. It is particularly important in commercial orchards of parts of West Virginia (5,

FIG. 131. Red cedar gall, showing telial horns.

6) and Virginia, New York (7, 15, 16), Wisconsin River valley of Wisconsin, and Iowa (2). The disease is not reported west of the Rocky Mountains. In Europe, *G. juniperinum* (L.) Mart. (*G. tremelloides* R. Hartig) is the common species on apple (8).

Damage to apple is brought about chiefly by premature defoliation. Sometimes fruits are affected and, since they become deformed, are not satisfactory for market (Fig. 130). On ornamental red cedars the production of galls and their unsightly appearance (Fig. 131), especially when telial horns are produced in the spring, are the chief causes of damage (3).

Symptoms. On apple the disease appears on leaves as small greenish-yellow spots which gradually enlarge, changing to orange yellow and becoming surrounded at the border by concentric red bands. Pycnia appear on the upper side of leaf lesions, and the yellowish exudate becomes conspicuous. On the lower side of the lesion hypertrophy follows, and eventually the aecia appear. The gymnosporangia have prominent peridia, and in apple rust they give a fuzzy appearance to the lower side of

the lesion. Lesions appear on immature fruits of apple, causing dwarfing and malformation (Fig. 130). Twigs of very susceptible varieties may be affected severely when infection early in the spring results in short stubby growth bearing pycnia and aecia. Seriously affected twigs may die by the end of the season.

On cedar the first sign is a minute greenish swelling on the upper (inner) surface of the leaf. It enlarges rapidly to form a brown gall, which may be up to an inch or more in diameter by autumn with many pit-like depressions over the surface. In warm moist spring weather long, pointed, golden-brown, jelly-like horns appear from the pits (Fig. 131). These are the telial sori and comprise the most conspicuous stage of the disease. The galls die later but remain attached to the tree up to a year or more.

The Causal Organism. *Gymnosporangium juniperi-virginianae* Schw., 1822.

Synonymy:

Gymnosporangium macropus Link, 1825.
Gymnosporangium virginianum Spreng., 1827.
Podisoma juniperi-virginianae Fr., 1832.
Caeoma (*Aecidium*) *pyratum* Schw., 1832.
Podisoma macropus Schw., 1832.
Aecidium pyratum Schw., 1832.
Roestalia penicillata Farlow, 1880.
Roestalia pyrata Thaxt., 1886.
Puccinia macropus Kuntze, 1898.
Puccinia juniperi-virginianae Arth., 1899.
Tremella juniperi-virginianae Arth., 1901.
Aecidium juniperi-virginianae Arth., 1905.

The aecia are commonly arranged in concentric rings. The fimbriate peridia are split to the base and curved backward when atmosphere is dry, allowing ready escape of aeciospores. When the lesion becomes wet, the peridia curve inward, closing the aecium (Fig. 132). The aeciospores are globoid or broadly ellipsoid, finely verrucose, 16 to 24 by 21 to 31 μ, with light chestnut brown walls. Pycnia are formed on the upper side of the leaf. Telia are cylindric or cylindric-acuminate, 1.5 to 3 mm. in diameter and 10 to 20 mm. long, arising from the cedar gall. Teliospores are two-celled, rhombic-oval or narrowly ellipsoid, 15 to 21 by 42 to 65 μ, pale cinnamon brown, pedicellate. The genus has been monographed by Kern (9).

Disease Cycle (2, 10, 11, 15). The fungus lives over as mycelium in the cedar gall. In the spring, when moist, moderately warm weather prevails, the telial horns are produced, and teliospores germinate in the gelatinous matrix to produce promycelia and sporidia (Fig. 133). The

latter are not discharged in any appreciable amount in temperatures below 10°C. (5). They are wind-borne and continue to be found in the sori over an extended period. Since there is no uredostage, no reinfection of cedar occurs, and sporidia infect only the apples. Penetration of the apple host is direct, the fungus invading the epidermal cell (Fig. 184) to form a primary hypha and proceeding inter- and intracellularly, forming small haustoria (13). The apple leaf is susceptible to penetration

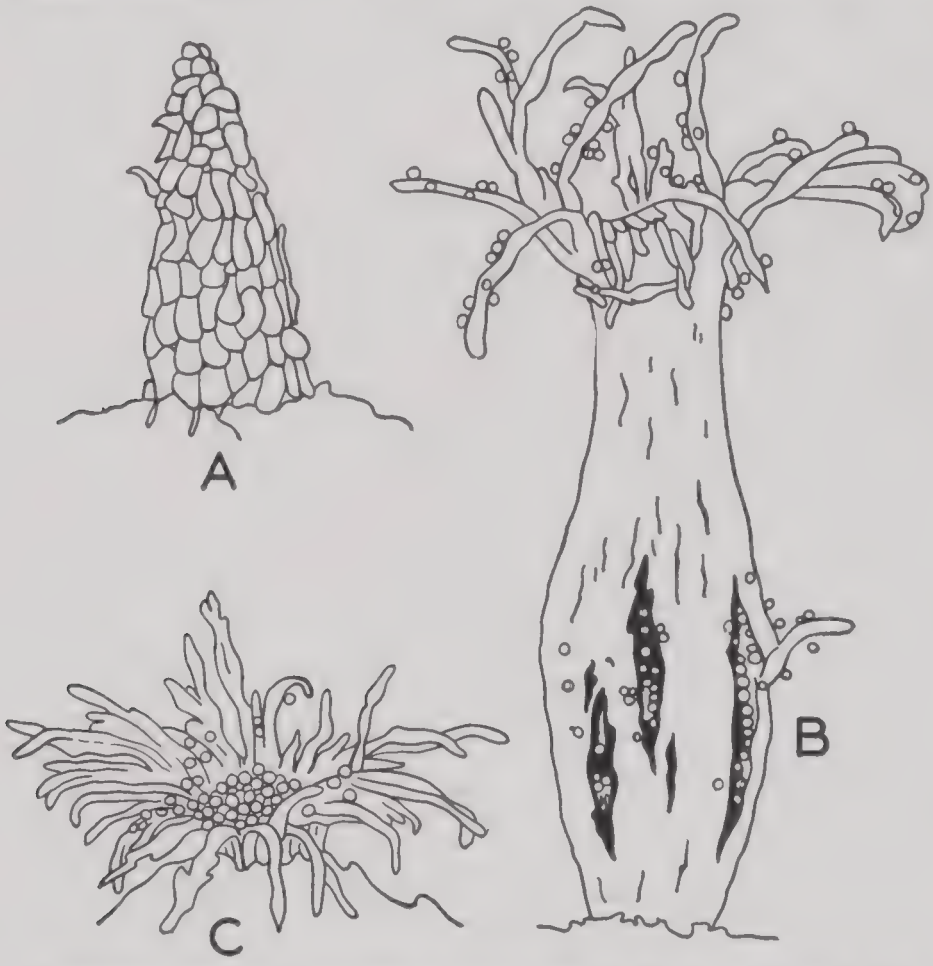

FIG. 132. *Gymnosporangium juniperi-virginianae*. *A*, the pointed tip of a young aecium which has pierced the epidermis of the leaf; *B*, side view of a mature aecium; strings of peridial cells are splitting downward, thus allowing the aeciospores to escape; *C*, aecium from which most of the spores have escaped. (*After Bliss.*)

only when young (5). Pycnia are formed, and pycniospores are exuded. Presumably the fungus is heterothallic, and mixture of exudates is provided by rain and insects. The binucleate stage is initiated in the aecial primordia, and aeciospores mature in August in northern states. Some difficulty is encountered in germinating aeciospores, and it is not clear whether they require a rest period and whether infection of cedar occurs in the fall or spring.

Initial gall development starts on the cedar leaf (Fig. 134) according to Weimer (16), who disagrees with Stewart's claim (14) that the galls

are transformed axillary buds. The galls consist chiefly of parenchyma with intercellular mycelium and haustoria of the fungus. Haustoria become extremely abundant just before telia form, and the starch, previously abundant, rapidly disappears, first in the region of the sori (14).

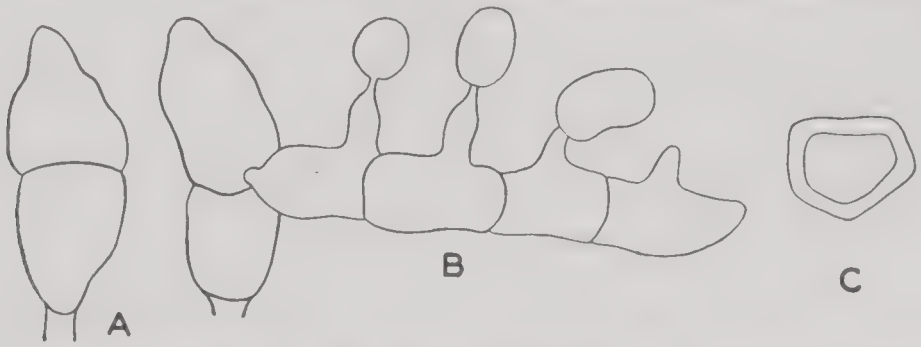

FIG. 133. *Gymnosporangium juniperi-virginianae*. *A*, teliospore; *B*, teliospore, promycelium, and sporidia; *C*, aeciospore. (*After Bliss.*)

Physiologic Specialization and Varietal Resistance. While relatively little work has been done on specialization, Crowell (3) and Bliss (2) both report evidence of physiologic races. Many resistant species

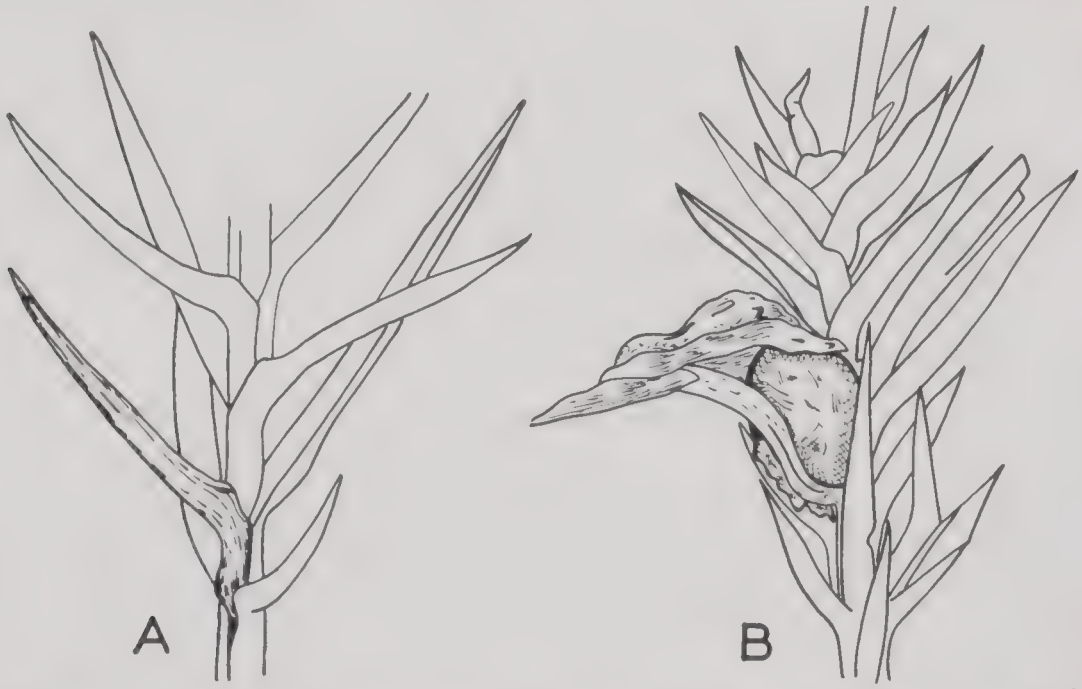

FIG. 134. Red cedar rust. *A*, early sign of infection on leaf which is chlorotic. Slight swelling has cracked the epidermis at the base of the leaf. *B*, a larger gall which has lifted the infected needle away from the stem. A strip of epidermis is still attached from the back of the needle to the base of the sheath. (*After Bliss.*)

of *Juniperus* are listed by Crowell (3). Many apple varieties are highly resistant, and numerous lists of apple varieties indicating relative resistance have been published (4, 6). While native red cedar and most ornamental varieties are susceptible, there is evidence that resistant varieties may be developed (1).

Moore (12) presented preliminary evidence that resistance in apple is monogenic and dominant over susceptibility. The relation of the pathogen to one susceptible and to three resistant apple varieties is described by Nusbaum (13).

Control. The most effective means of control is the choice of resistant apple varieties in planting of orchards where red cedar prevails. In some intensive commercial areas the removal of cedars within a mile of commercial orchards has at times been advocated, but this measure and the removal of galls are usually impractical. West Virginia has a law passed in 1912 compelling removal of red cedar within 3 miles of commercial apple orchards. Spraying has not been particularly successful because of the number and frequency of applications needed (5). Hamilton (7), however, is of the opinion that fungicidal control is practical in the Hudson valley. It is necessary to supplement the usual scab-control program by increasing the number of summer sprays of a wettable sulfur so as to keep new growth fully covered.

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FLAX RUST

In most regions where flax is grown intensively rust is present and is often a major disease. In the United States it is prevalent and particularly important in the Red River valley. The disease is reported to have

Fig. 135. Flax rust. (*After Dickson.*)

reduced the flaxseed crop in the north central states in 1942 by 5 million bushels. The disease is also injurious to the fiber in flax grown for this purpose in Europe and in Canada. The host range of the pathogen extends to about a dozen species of *Linum*, but subspecies specialized to various host species occur.

Symptoms. The pycnia are relatively inconspicuous; the first signs of the disease are the orange-yellow aecia which appear on leaves and stems (Fig. 135). Later the reddish-yellow uredia on stems, leaves, and capsules become the conspicuous phase of the disease. Chlorotic zones may surround the sori. The telia are most common on the stem, giving a dark to black appearance to the sori in the last part of the growing season.

The effect upon the plant is to reduce vigor and yield and induce breaking of the stem.

The Causal Organism. *Melampsora lini* (Pers.) Lév., 1847.

Synonymy:
Uredo miniata β lini, Pers., 1801.
Xyloma lini Ehr., 1818.
Melampsora liniperda Palm, 1910.

The full life cycle was established by Arthur (2) in 1907. This species is autoecious. The pycnia are subepidermal, forming first near the upper and later near the lower side of the leaf. Growth of the pycnium centers beneath a stoma, the latter serving as an ostiole. The orange-yellow aecia are most abundant on the lower side of the leaf. The aeciospores are globose, 19 to 27 by 21 to 28 μ, and finely verrucose. The reddish-yellow uredia occur on both sides of the leaf and on the stem. Urediospores are broadly ellipsoid or obovate, 13 to 20 by 15 to 25 μ, and finely verrucose. The telia are subepidermal, brown when fresh, turning black with age. As in many members of the Melampsoraceae, the teliospores are one-celled and closely packed in a layer. They are prismatic, brown, 10 to 20 by 42 to 59 μ.

Disease Cycle. The fungus overwinters in temperate areas as teliospores. In regions where summer and winter flax crops are grown, the fungus may be perpetuated by the uredial stage. Teliospores require a rest period of several months. They germinate in the spring to form promycelia and haploid sporidia. The germ tubes of sporidia penetrate the host directly, being at first intracellular and later primarily intercellular. Pycnia are formed, and pycniospores and paraphyses emerge from the pycnium through the ostiole (Fig. 136). Of the numerous rusts studied cytologically this is the only species in which haploid mycelia do not commonly protrude to the surface of the leaf through the epidermis or through stomata (1). The fungus is heterothallic. According to Allen (1) pycniospores probably invade through compatible pycnia or through the host epidermis. When the invading haploid mycelium contacts compatible hyphae or pycniospores, fusion follows. The internal mycelium next becomes binucleate, and aecia and aeciospores form.

Aeciospores and urediospores do not germinate in a saturated atmosphere unless they are in contact with liquid water. They germinate over a temperature range from slightly above 0°C. to about 27° with an optimum at about 18° (5). Spores in uredia remain viable up to 7 weeks at 7° and 20 to 80 per cent relative humidity, but at 22° very little germination occurs after 3 weeks. Infection occurs at from 7 to 26° with the optimum at 16 to 22° (5). Misra (8) reported optimum field development at about

20°C., but there was marked suppression of infection when the temperature reached about 30°C. for 2 or 3 days. Urediospore germ tubes grow toward stomata and produce appressoria over the stomatal openings. A slender penetration tube arising from the appressorium enters the stoma and produces a substomatal vesicle, from which infection threads grow into the intercellular spaces and send haustoria into the host cells. The mycelium is most abundant in the parenchyma tissue, but it also invades the fibers. Invasion of tissue is enhanced by luxuriant growth of the host (5).

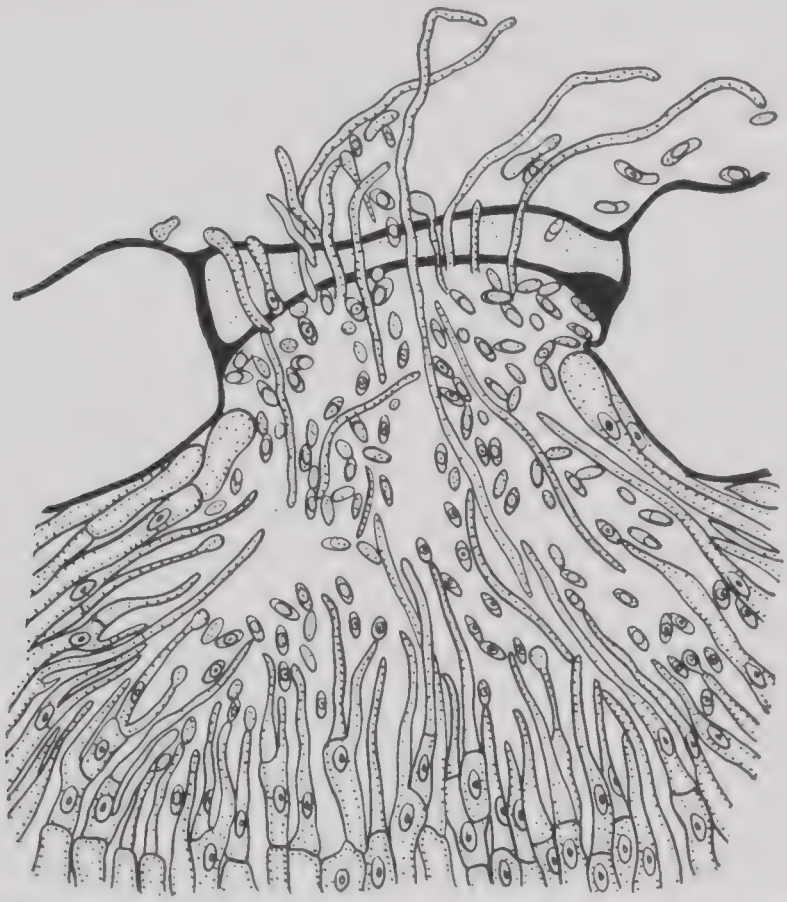

FIG. 136. *Melampsora lini*. Upper part of a pycnium, showing the stoma of the host to be serving as an ostiole. (*After Allen.*)

Physiologic Specialization and Varietal Resistance. According to Hart (5), Körnicke suggested as early as 1865 that the rust on common flax (*Linum usitatissimum* L.) was distinct from that on *L. catharticum* L., since in Germany the latter was commonly rusted when the former remained free. Eriksson in 1915 regarded the two forms as distinct. Hart found that in Minnesota the form on common flax and that on wild flax (*L. lewisii* Pursh) were distinct. Numerous physiologic races of *Melampsora lini* are now catalogued. It appears that the groups of races which exist in the United States, Argentina, Europe, and Australia are quite different. Since the rust is autoecious and aecia are relatively common, it might be

expected that continual hybridization within races of a locality would result in all possible combinations being formed. That this is not necessarily the case is shown by the work of Flor (3) in the Red River valley; he crossed two races and in the F_2 found many new races among the segregates.

Henry (6, 7) in 1926 reported Ottawa 770B, Argentine, and Bombay varieties immune to several collections of rust in the United States. In crosses with susceptible varieties he found resistance in Ottawa and Bombay dependent on a single dominant gene; that in Argentine was apparently dependent on two gene pairs. Myers (9) in a study of more varieties with additional collections explained his results by assuming genes for resistance in two allelic series. Sharvelle (10) found that varieties could be placed in five more or less distinct groups on the basis of their reaction to a given race, the classification following closely the pattern set up for black stem rust (p. 411). He also studied various biochemical, physiological, and morphological factors which were associated with resistance.

In a still later study with 20 flax varieties, 16 of which were used as race differentials, Flor (4) defined at least 19 pairs of genes as conditioning response to the fungus. Sixteen of these lie in three linkage groups. In two of the groups the genes are considered allelomorphic while in the third group they are closely linked. In a study of the genetics of pathogenicity of the fungus it was indicated that for each pair of genes conditioning rust reaction in the host there is a corresponding pair in the pathogen.

Control. Fragments of capsules containing telia may contaminate the seed. Thorough cleaning of seed is essential when the crop is to be planted on land new to flax. The major control is through the development of resistant varieties. Breeding programs for this purpose are under way in various parts of the world. In 1945, six new rust-resistant varieties were in use in the flaxseed region of the north central states. These varieties developed by the U.S. Department of Agriculture in cooperation with the experiment stations in Minnesota, North Dakota, and Montana are Arrow, Crystal, Dakota, Renew, Sheyenne, and Victory. They were resistant to all races found in that region at that time. The greatest danger of their decline lies in the possible introduction of races from South America to which they are not resistant. Dakota was later found to be susceptible to a new race which appeared in the Red River valley.

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Fig. 137. White-pine blister rust. (*Courtesy of U.S. Department of Agriculture.*)

WHITE-PINE BLISTER RUST

Stands of five-needled pines in North America have been jeopardized by this disease since early in the twentieth century. The introduction of the pathogen into the United States and its gradual spread throughout the northern part of this country and in Canada will be discussed in Chap. 15. The pathogen alternates between species of *Pinus* and species of *Ribes*, including wild and cultivated forms of currant and gooseberry. The fungus was first found with certainty by Dietrich in 1854 in the Baltic region. The two stages were found, one on *Ribes* and the other on *Pinus strobus* L., but the connection between them was not established until the work of Klebahn in Germany in 1905. It is probable that *P. cembra* L., which ranges across northern Asia, was the original pine host. The disease has been destructive on five-needled pines in western Europe (17). It was discovered in North America at Geneva, New York, in 1906. It is now widespread throughout the range of white pine in northeastern United States and southeastern Canada and in the white-pine forests of British Columbia, Washington, Idaho, Montana, Oregon, and California.

Many species of *Ribes* are known to be susceptible to the fungus. Spaulding (21) listed 11 susceptible species of white pines, *i.e.*, *P. koraiensis* Sieb. & Zucc., *P. cembra* L., *P. flexilis* James, *P. lambertiana* Dougl., *P. parviflora* Sieb. & Zucc., *P. peuce* Grieseb., *P. excelsa* Wall., *P. monticola* Dougl., *P. strobus*, and *P. aristota*. Bedwell and Childs (2) added *P. albicaulis* to the list of susceptible species.

A complete historical discussion and a summary of investigations of the disease up to about 1920 are given by Spaulding (21).

Symptoms. The disease appears on pine first on the needle as tiny yellow spots centering on the line of stomata on the under side of the leaf. The spots turn golden yellow, producing a yellow mottle on infected leaves. As the fungus invades the twig, the latter also becomes yellow. Invasion of branches up to 3 to 4 years old occurs from infected twigs (Fig. 137). They enlarge to produce spindle-like swellings. Cankers on branches and trunks continue to enlarge from year to year. The invaded bark first becomes yellow or bronzed. The pycnia appear first in the center of the lesion, bearing exudate containing pycniospores. Later, aecia are produced in place of the pycnia, and new pycnia appear in an outer zone. After the aecia have shed aeciospores, the bark tissue dies. In an advancing canker there is, then, beginning at the outside, a yellow zone showing swelling, a zone showing pycnia, a zone bearing aecia, and an inner area of dead bark where aecia have formed in previous seasons (Fig. 138). The aecia appear as raised blisters in the spring. They begin to push through the bark several weeks before they rift the host surface and discharge yellow dusty masses of aeciospores. Resinous extrusions from the lesion may occur.

On *Ribes* the uredia appear as slightly raised yellow spots, chiefly on the lower side of the cotyle-

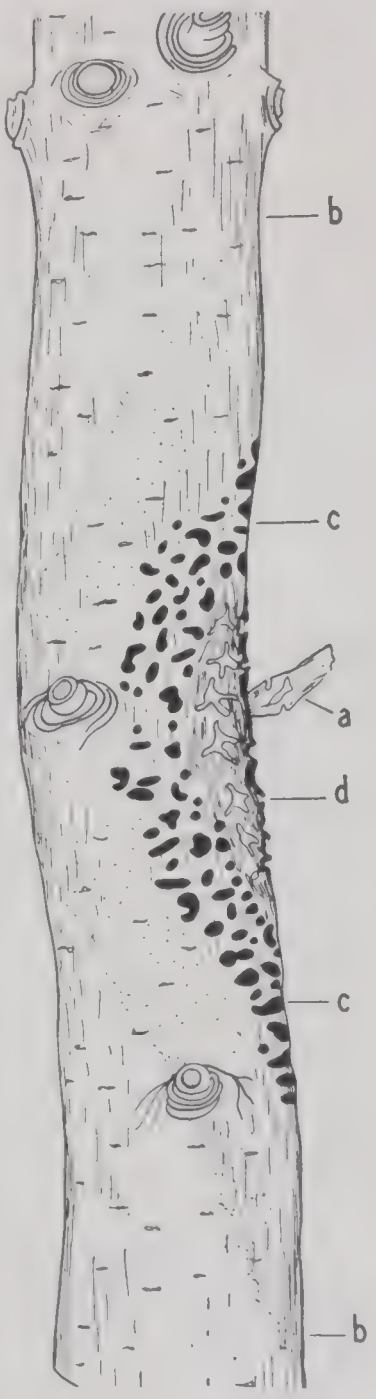

FIG. 138. White-pine blister rust. Drawing of an infected 12-year-old main stem. The fungus entered along a small branch at *a*. The advancing edge of the lesion is shown at *b*. The pycnial area is shown at *c*. The aecial area in which the bark is cracked and broken is at *d*. In another year the aecial area would spread over the present pycnial area; the pycnial area would advance to the present boundary; the boundary would advance accordingly. (*After Colley.*)

dons, leaves, floral bracts, bud scales, petioles, and young stems. The uredia and telia vary in size and arrangement with the species or variety of the host. In some the sori are small. In others they are large and closely crowded. The telia which follow uredia are slightly darker, and

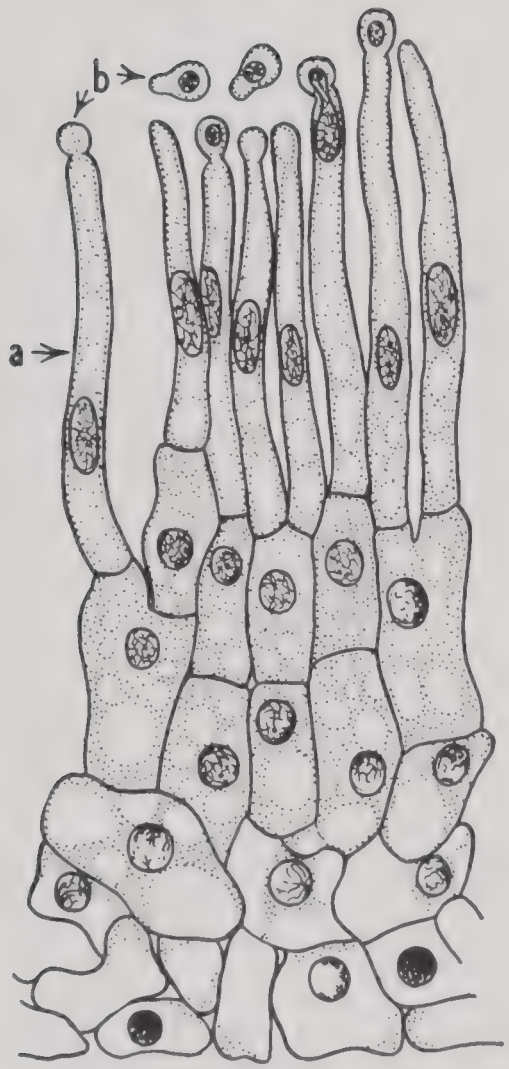

Fig. 139.

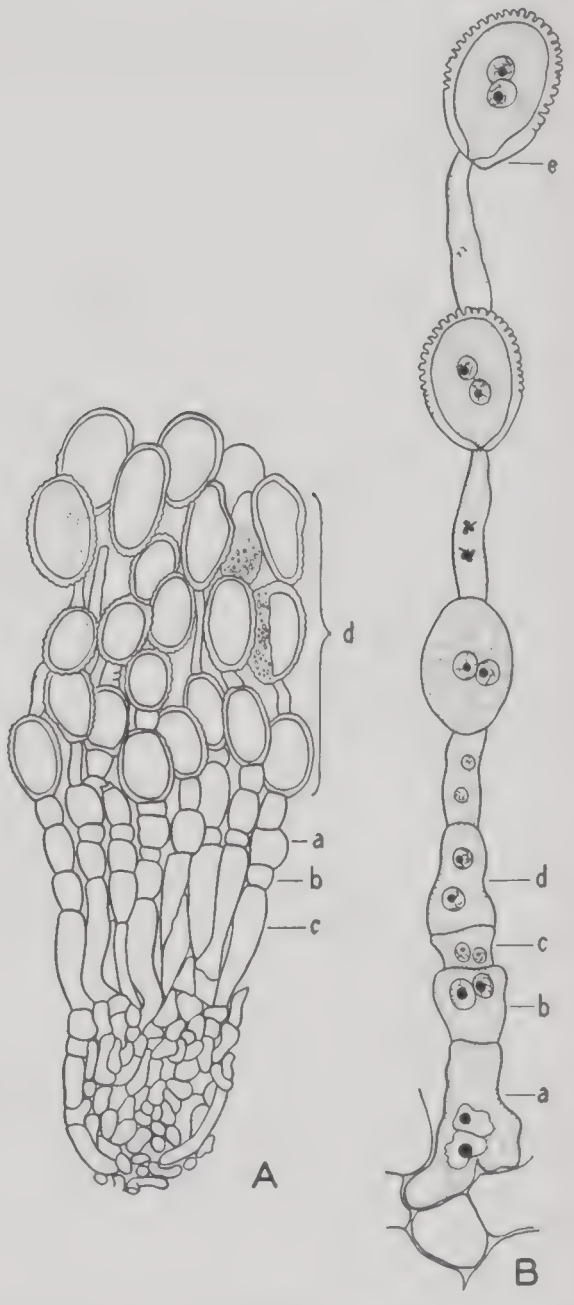

Fig. 140.

Fig. 139. *Cronartium ribicola*. The elements of a pycnium. The cells at the base are almost empty. Above them are the short branching trunks which bear the sporophores *a*; *b*, young and mature pycniospores. (*After Colley.*)

Fig. 140. *Cronartium ribicola*. *A*, section through the edge of an aecium: *a*, young aeciospore; *b*, intercalary cell; *c*, basal cell of spore chain; *d*, potential aeciospores becoming modified into peridial cells. *B*, aeciospore chain: *a*, basal cell; *b*, aeciospore initial; *c* intercalary cell; *d*, young aeciospore; *e*, mature aeciospore. (*After Colley.*)

the teliospores are borne in columns up to 2 mm. in height. Some host varieties defoliate prematurely following infection.

The Causal Organism. *Cronartium ribicola* J. R. Fischer, 1872.

Synonymy:

Cronartium ribicola Dietr., 1856.
Peridermium pini f. *corticola* Dietr., 1856.
Peridermium strobi Kleb., 1887.

Pycnia are formed within the cortex of the stem, causing small blisters (Fig. 139). The aecia form on globoid galls rupturing the peridium.

Fig. 141. *Cronartium ribicola*. Median section through a small mature uredium on *Ribes*. (*After Colley.*)

Aeciospores are ellipsoid, 18 to 20 by 22 to 31 μ, coarsely verrucose (Fig. 140). The urediospores are ellipsoid or obovoid, 14 to 22 by 19 to 35 μ, sharply echinulate (Fig. 141). The teliospores are oblong or cylindric, 8 to 12 by 30 to 60 μ, rounded or obtuse at both ends (Figs. 142, 143).

Disease Cycle. The fungus may overwinter as mycelium in stems of *Ribes*. It also overwinters as mycelium in infected pine. Aecia form in the spring, the time of maturity varying with climatic conditions. They may open by the first week of April in northern states and continue to discharge aeciospores for about 4 weeks. The late aeciospores may occasionally remain viable over winter. As a rule they are viable for about 4 weeks. The aeciospores are wind-borne and may be carried up to 7 miles. Transmission by gypsy moth larvae has been demonstrated. The aeciospores germinate over a range of 5 to 19°C. with an optimum of 12°. Infection

occurs through stomata. The *Ribes* leaves must be moist for infection to occur. Susceptibility decreases with age of the leaf. Uredia may appear in as short an incubation period as 7 days. Several generations of uredia may form during the summer season. Urediospores have been caught in the germinable condition 3,200 ft. from their origin. Wind is the chief disseminating agent. The spores germinate at from 8 to 25°C. with an optimum of 15°. They have been found to remain viable up to 270 days.

FIG. 142. *Cronartium ribicola*. Mature telial column on *Ribes* in which teliospores have germinated to produce promycelia and sporidia. (*After Colley.*)

The teliospores are not readily detached from the column. They germinate without a rest period and produce sporidia in the fall. Sporidia have been trapped 600 ft. from their source. In the main about 1000 ft. is regarded as a practical safe distance between pine and *Ribes* to protect the former from sporidial infection. Germ tubes from sporidia enter stomata of pine leaves and form substomatal vesicles. The mycelium enters the vascular system and grows rapidly down the twig. Hirt (9) found the minimum time for infection under optimum conditions to be about 11 hr. The optimum temperature for infection is 10 to 18°C. Liquid water on the leaves is essential. Most of the infection occurs in one or two relatively brief, cool, wet periods after teliospores are mature.

Varietal Resistance. There is as yet no information on heterothallism and physiologic specialization in this fungus. Species of white pine vary in resistance. *Pinus strobus*, *P. monticola*, and *P. albicaulis* are very susceptible. *P. cembra* has two subspecies one of which (*P. cembra helvetica*), native to Switzerland, is highly resistant. *P. peuce* and *P. excelsa* are also highly resistant. Occasional highly resistant trees in *P. strobus* have been found. The possibility of propagating vegetative clones from such trees is being explored (19).

A wide range in resistance also occurs in *Ribes*. *R. nigrum* L. is the most susceptible species. Red currants are susceptible but are regarded by some investigators as not so dangerous as alternate hosts as some other forms of *Ribes* because of the paucity of telia and early leaf cast. Several varieties of red currant within the species *R. petraeum* are highly resistant or immune (6). A staminate clone of *R. alpinum* L. was immune while the pistillate clone was susceptible (5). A clone of immune black currant (*R. ussuriense*

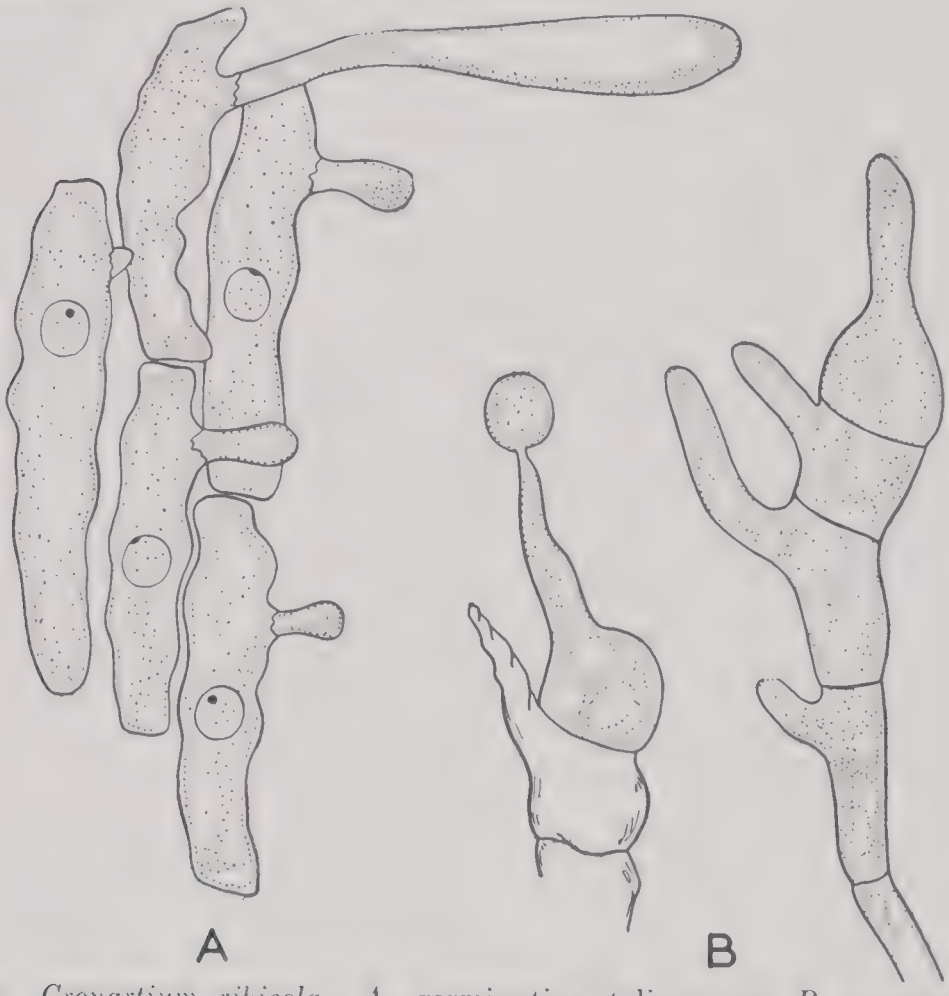

FIG. 143. *Cronartium ribicola*. *A*, germinating teliospores; *B*, promycelia and sporidium. (*After Colley.*)

L. crossed with *R. nigrum*) has been reported (7). The interaction between the pathogen and Viking, a resistant red currant, is discussed by Anderson (1). The Viking has remained highly resistant in widespread trials (8).

Control. The most important means of control of the disease on white pine is eradication of the *Ribes* host. This is particularly important where new stands are being established. The program of eradication in the United States is discussed in Chapter 15. Various methods and materials for eradication of *Ribes* are discussed by Offord *et al.* (18).

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RHIZOCTONIA DISEASES

Late in the eighteenth century mycologists in Europe encountered a fungus on the saffron flower, *Crocus sativus* L., which was conspicuous by the presence of sclerotioid mats on the host substrate. Persoon in 1801,

regarding the fungus as a sterile form, placed it in the genus *Sclerotium*. De Candolle in 1815 created the genus *Rhizoctonia* to accommodate the fungus on crocus, *Rhizoctonia crocorum* (Pers.) DC., and allied forms on alfalfa, *R. medicaginis* DC., and apple, *R. mali* DC. The Tulasnes published the first extensive morphological study of the genus in 1862; they regarded the forms on saffron and alfalfa as belonging to the same species. The disease referred to by the above-mentioned writers is that now known as the violet root rot. This disease occurs on many species of plants in widely separated parts of the world, and it is fairly readily distinguished by its distinctive symptoms.

In 1858, Kühn published in his textbook a description of another *Rhizoctonia* disease on potato. He considered the disease distinct from the violet root rot and described the fungus as *Rhizoctonia solani* Kühn. The latter species also has a very wide host range. Much confusion between the two diseases and pathogens in the literature followed from 1858 to 1915. This was because of lack of authentic type specimens and because of the absence of knowledge regarding spore forms in either species. A redefinition of the two species was published in 1915 by Duggar (10), who had taken the opportunity to confer on the subject with Kühn in Germany in 1899.

The two diseases on the various hosts are referred to herein as violet root rot and common *Rhizoctonia* disease, respectively. Violet root rot was first recorded in this country in 1890 in Nebraska on alfalfa. It was reported on potato in Oregon in 1915. It has since then been found from time to time on alfalfa, potato, and sugar beet but is relatively rare and spasmodic in its occurrence.

The common *Rhizoctonia* disease is world-wide in distribution and affects many species of plants. The fungus attacks the host at one or more stages in the development of the latter with the result that more than one distinct disease has become differentiated on a single species. A range of common names is to be found in the literature referring to one or more phases of the disease, *e.g.*, damping-off of cotton, sore shin of cotton, wire stem of cabbage, *Rhizoctonia* head rot of cabbage, black root of beet, crown rot of beet, black scurf of potato, stem canker of potato, aerial tuber and top rosette of potato, etc.

The relation of *R. solani* to the Basidiomycetes was not established until early in the twentieth century. The basidial stage is, in fact, an inconspicuous saprophytic stage of the pathogen. Patouillard in France was the first to describe the basidial stage as *Hypochnus filamentosus* in 1891, and Prillieux and Delacroix described it, in the same year and country, on potato stems as *Hypochnus solani*. None of the investigators suspected a connection with *R. solani*, however. The fungus was found on potato stems in Colorado in 1903 by Rolfs and the material was referred by Burt to

Corticium vagum Berk. & Curt., a saprophytic fungus described in 1873. Because of the parasitic nature of the potato form, Rolfs designated it as *C. vagum* Berk. & Curt. var. *solani* Burt. He germinated basidiospores and secured a fungus identical with *R. solani*. In 1943, Rogers (30) transferred the fungus to the genus *Pellicularia*. In 1927, Buddin and Wakefield (5, 6) demonstrated that a previously described Basidiomycete, *Helicobasidium purpureum* (Tul.) Pat., is the basidial stage of *R. crocorum*, the violet-root-rot fungus.

Symptoms. *Violet Root Rot.* The above-ground symptoms are not distinctive. The foliage often is chlorotic; the plants may wilt and die rather suddenly. The disease is usually localized, and, in the late stages, areas of dead plants in the field are striking. When affected plants are pulled, the diseased subterranean portions show almost unmistakable signs. The superficial mat of the fungus is pale buff to violaceous in the early stages, turning to red violet or violet brown as the infection advances. The dense fungus mat contains numerous closely aggregated papillae, which are sclerotial-like mycelial bodies, slightly darker than the rest of the mycelial growth.

Common Rhizoctonia Disease. Four phases of the disease are (*a*) damping-off; (*b*) stem canker and root rot; (*c*) storage-organ decay; (*d*) foliage blight or spot.

Damping-off is a disease of young seedlings in which the rapidly expanding plant consists preponderantly of thin-walled parenchyma and relatively few lignified or suberized cell walls. Under conditions favorable to *R. solani*, infection of hypocotyl and root is followed by rapid collapse of plants before or shortly after emergence above ground. The disease is not ordinarily distinguishable from damping-off incited by phycomycetous pathogens, particularly species of *Aphanomyces* (p. 183), *Pythium* (p. 188), and *Phytophthora* (p. 197). There may be a combined effect of Phycomycete and *R. solani* in which one or the other may be the forerunner. Many dicotyledons and occasionally a monocotyledon, such as onion, may be affected.

Stem canker and **root rot** are expressed in various forms depending upon the host and the environment. Potato is one of the most interesting and important cases (Fig. 144). When seed tubers sprout, the growing tip is particularly susceptible to *R. solani*, and under some conditions it may be killed before emergence. In such a case a dormant bud near the base of the sprout grows out; either it is killed, or, if conditions change, it may emerge normally. Early irregularity in potato stands is often due to the persistent killing of young sprouts until the environment changes sufficiently in favor of the host. Ordinarily as plant stems and roots become established and differentiation of tissues gets under way, the host becomes more

resistant and the lesions more circumscribed and less injurious. Stem cankers on older stems of potato, bean, and other plants are shallow and brownish. Cabbage (42) and stock (9) seedlings pass through a very susceptible stage, when damping-off is common, to a stem-canker stage (wire stem), when the injury is confined chiefly to the sloughing primary cortex or to the outer layers of the periderm. A similar shift from damping-off to stem canker (sore shin) is common in cotton. Brown patch of turf is a phase in which the fungus is very active during a period of favorable environment, killing leaves and stems but allowing tops to recover by new growth when the conditions are less favorable for the pathogen.

The secondary effects of stem and root injury are various, and they may constitute the most conspicuous and destructive phase of the disease. When stem cankers are pronounced on potato, normal downward flow of carbohydrates to the tubers is disturbed, and their accumulation in the tops is accompanied by stunting, rosetting, enhancement of purpling due to excessive anthocyanin formation, and formation of aerial tubers in axes of branches and petioles.

The expression of the **storage-organ phase** varies with host and environment. Potato tubers are affected superficially by the development of brown to black sclerotia closely appressed on the skins (Fig. 144). Only rarely does the pathogen invade the tuber tissue to cause any breakdown (26, 38). On the fleshy roots of other plants decay may be more destructive. A semisoft rot occurs in roots of radish, turnip, and carrot. Sugar beet is infected late in the growing season, the fungus becoming established in the bases of the leaves and progressing into the fleshy root. A brown to black dry rot follows, and deep cracks appear at the surface of the diseased root. Bean pods, when affected, show dark brown sunken lesions with well-defined borders. In immature tomato fruits the decay is semisoft and brown, and the surface of the decayed area is marked by concentric rings. When cabbage and head lettuce are affected in the field after midseason, "bottom rot" appears first. The lower outer leaves are affected by the fungus advancing from the main stem along the petiole and midrib, causing dark lesions and gradually killing the leaves (39). The fungus invades cabbage heads and overruns the surface leaves to cause dark sunken spots and a slow but destructive decay (head rot) (42, 44).

Leaf blight incited by *R. solani* is relatively uncommon. Brown patch of turf grasses is an exception. Kotila (16) described a strain of the organism which is responsible for a severe leaf blight of sugar beets. Heart leaves are reduced to tipburned stubs and blades of older leaves may become necrotic in part. A foliage blight (web blight) on lima bean, snap bean, and numerous other hosts described in Florida is incited by *Corticium microsclerotia* Weber (43). A leaf spot of citrus in Surinam was de-

scribed by Stahel as incited by *C. arcolatum* Stahel (36). Rogers (30) has reduced the last two species to synonymy with the common *Rhizoctonia* disease pathogen, *Pellicularia filamentosa* (Pat.) Rogers.

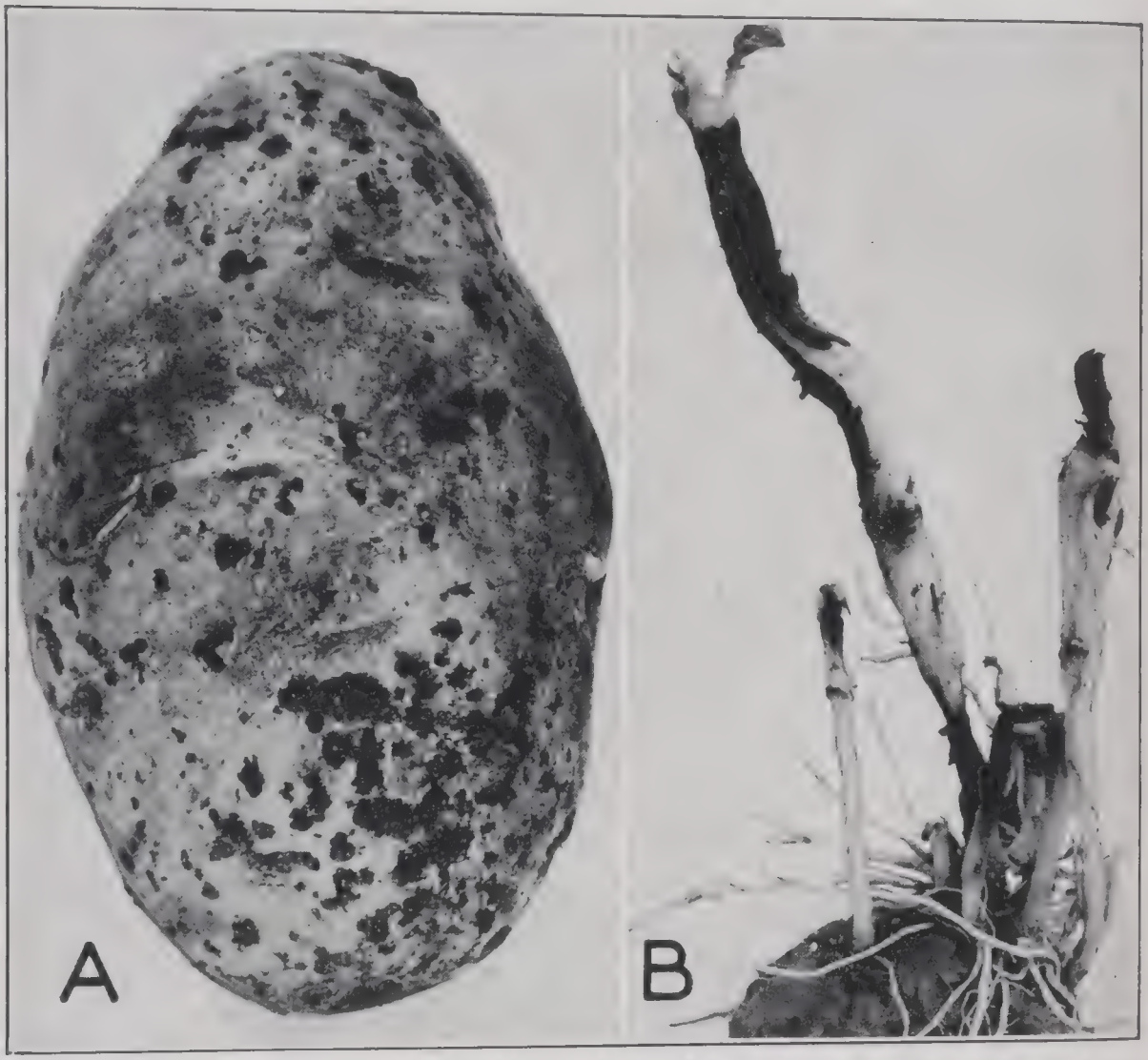

FIG. 144. *Rhizoctonia* disease of potato. *A*, black scurf stage on the tuber; *B*, stem lesions on shoots rising from a seed piece. Note that as shoots are killed back at the tips, younger shoots are rising from the eye.

The Causal Organisms.

1. Violet-root-rot fungus. *Helicobasidium purpureum* (Tul.) Pat., 1885.

SYNONYMY:

Mycelial stage:

Tuber parasiticum Bull., 1791.
Sclerotium crocorum Pers., 1801.
Rhizoctonia crocorum DC., 1815.
Rhizoctonia medicaginis DC., 1815.
Thanatophytum crocorum Nees, 1816.
Tuber croci Duby, 1830.

Rhizoctonia rubiae Dcne., 1837.
Rhizoctonia dauci Rab., 1859.
Rhizoctonia violacea Tul., 1862.
Rhizoctonia asparagi Fckl., 1869.
Basidial stage: *Hypochnus purpureus* Tul., 1865.

The young hyphae are dilutely violaceous with a pigment which may be decolorized by acidulated water. The hyphal branches arise at right angles with a septum not more than 10 μ from the junction. The mycelium is branched, septate, distributed uniformly over the host substrate, and sometimes aggregated into strands which are macroscopically visible. Deep violet brown sclerotia form on the larger strands, either on the host root or in the soil. They are flattened or rounded, covered with a thick velvety felt, and vary in diameter from a few millimeters to several centimeters. Small stromatoid aggregates of mycelium which form on the host surface serve as infection cushions from which hyphae penetrate the root cortex.

Duggar (10, 11) and earlier workers did not succeed in growing the fungus in pure culture. Diehl (8) in 1916 was the first to report success in this direction with the use of "radicicola" agar containing only sucrose and dipotassium phosphate, on which the fungus grew very slowly. In 1917 Van der Lek (40) was successful with malt agar. Buddin and Wakefield (4) showed in 1924 that the fungus, when isolated completely from contaminating fungi and bacteria, would grow on a wide range of artificial media. The last-named investigators found the basidial stage to occur only during a very limited period in the spring in England, when it is present most often on the surfaces of bases of herbaceous plants near the soil. The felt-like hymenium is purplish or violet in color. The basidium is hyaline, the elongating apex gradually bending over while septa appear. Sterigmata arising from each cell (two to four) vary in length from 10 to 35 μ and bear hyaline, ovate, elliptic, oblong, or reniform basidiospores (10 to 12 by 6 to 7 μ). Conidia are reported in pure culture by Buddin and Wakefield (5).

2. Common *Rhizoctonia* disease. *Pellicularia filamentosa* (Pat.) Rogers, 1943.

Synonymy:
Mycelial stage:
Rhizoctonia napaeae West., 1846.
Rhizoctonia rapae West., 1852.
Rhizoctonia solani Kühn, 1858.
Rhizoctonia betae Eidam, 1887.
Basidial stage:
Hypochnus filamentosus Pat., 1891.
Hypochnus solani Prill. & Del., 1891.

Corticium vagum Berk. & Curt. var. *solani* Burt, 1903.
Corticium botryosum Bres., 1903.
Corticium solani (Prill. & Del.) Bourd. & Galz., 1911.
Botryobasidium solani (Prill. & Del.) Donk., 1931.
Corticium microsclerotia Weber, 1939.
Corticium areolatum Stahel, 1940.

The fungus grows well on culture media. The mycelium is colorless when young, becoming brown when old, occurring in visible brown strands on the host substrate. Branches when young are inclined in the direction

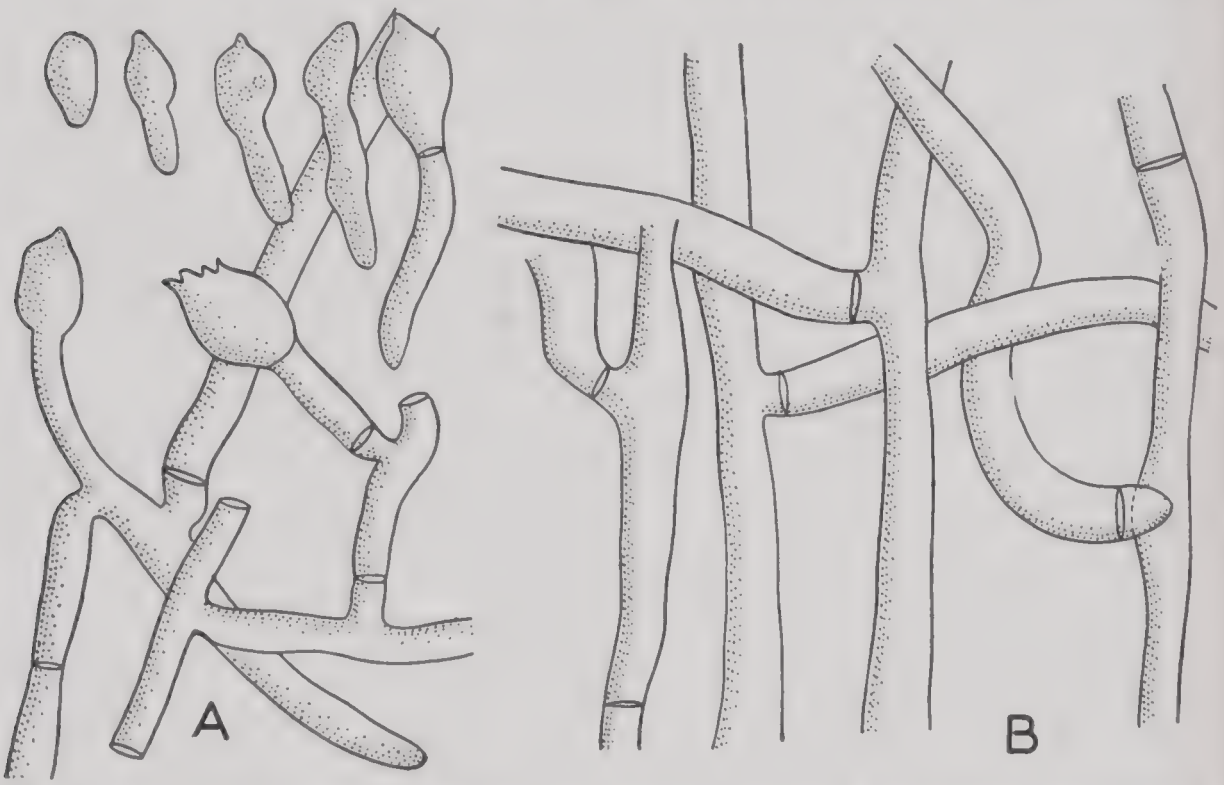

FIG. 145. *Pellicularia filamentosa*. *A*, basidia, one with sterigmata; germinating basidiospores; *B*, vegetative mycelium. (*After Harter and Weimer.*)

of growth and are constricted at the junction with the main hypha, but as they grow older, they assume a right-angle relation to the latter (Fig. 145). Under certain conditions and on certain substrates the mycelium becomes tufted, dividing into short ovate cells and eventually developing into brown sclerotia.

The basidial stage appears as a flaky pellicle on the surface of host stems or leaves near the soil under conditions of high relative humidity. The basidia, borne on small imperfectly symmetrical cymes, are barrel-shaped, obpyriform, or clavate (12 to 18 by 8 to 11 μ), bearing four sterigmata which arise as blunt knobs and later become horn-shaped (5.5 to 12 by 1.5 to 3.5 μ). Basidiospores are ellipsoid or oblong-ellipsoid, flattened on one side, 7 to 12.5 by 4 to 7 μ (Fig. 145). Müller (22) considered the

fungus homothallic, but wide variation in isolates from single basidiospores (13) indicates that considerable hybridization occurs. The thallus is multinucleate, becoming binucleate in the hymenium. Fusion of two nuclei occurs in the basidium, and meiosis follows to form four nuclei each of which passes into a basidiospore. Division in the spore results in most mature basidiospores being binucleate.

Disease Cycle. The remarks here will be confined to common *Rhizoctonia* disease, since little research has been carried out with violet root rot. The fungus lives from season to season in the soil or as sclerotia on propagative plant parts, *e.g.*, potato tubers. Elmer (12) found that the fungus did not survive in the soil during dry hot midsummer in Kansas. Baker (2) has shown it to be seed-borne, the fungus invading the seeds of pepper, eggplant, tomato, and *Linnia*. Townsend (39) reported penetration of lettuce leaf directly through the cuticle or through stomata.

The fungus has an optimum temperature on culture media of 25 to 30°C. with a minimum of about 8° and maximum of 31 to 35° (1, 17, 20, 22, 27, 31, 41, 44). Sclerotia germinate over a range of 8 to 30° with an optimum at 23°; the optimum for germination of basidiospores is 21 to 25° (22). Richards (27–29) studied the relation of temperature to development of stem lesions on potato, pea, and bean. In each case the optimum temperature was about 18°. Hemmi (14) found the fungus most active at 16 to 24° in damping-off of garden cress. Yu (46) reported the optimum on broad bean as 16 to 20°. Walker (41) reported sore shin on cotton most severe at 17 to 23°. Roth *et al.* (31, 32) stated the optimum for postemergence damping-off of red-pine seedlings to be about 28°.

Soil moisture is regarded by most workers as secondary to soil temperature in influence on stem canker. When the fungus is invading the dormant storage organs rather than the growing tissues of the plant, the optimum temperature is closer to that of the organism on artificial media. Thus while Richards (29) found the optimum on growing bean stems to be 18°, Lauritzen *et al.* (18) found the optimum on bean pods in transit to be 24° or above. Wellman (44) found the optimum for head rot of cabbage to be 23 to 27°. Lauritzen (17) reported the optimum for turnip rot at about 25°.

Physiologic Races. Many investigations have shown differences in cultural characters and pathogenicity of isolates of *R. solani* (13, 15, 17, 19, 20, 23–25, 33, 34, 37, 42, 44). There is abundant evidence of host specialization, but the degree of specificity is variable. The isolates from sclerotia on potato tubers are often low in virulence compared with isolates from potato stem cankers (25, 33). Isolates from potato are sometimes nonpathogenic on cabbage (44) and sugar beet (20), while isolates from sugar beet may be severely pathogenic on potato (20, 34). Although the fungus

is omnivorous and subsists saprophytically in the soil, there is evidence that rotation of a given crop with other crops may tend to reduce the strains pathogenic on that crop. This was shown in New York for potato by Blodgett (3) and for lettuce by Townsend (39). LeClerg (19) suggests that in potato sugar-beet rotations less damage to each crop may be expected if beets follow potatoes than if the reverse order is used.

Control. The damping-off stage, when it occurs in greenhouses, hotbeds, cold frames, and seedling nurseries, is controlled by soil treatment (Chap. 16). In the field, rotation, as indicated above, is of some value in special situations.

Inasmuch as the host usually grows into a resistant condition relatively rapidly after the early seedling stage, various means have been devised to protect the host or retard the pathogen during this period. A mercuric chloride drench applied along the row at the bases of young cabbage seedlings was worked out in New York State, but this measure has not come into general use. Providing best possible tilth for seeding is of importance because it encourages prompt growth. When the nature of the soil permits, shallow planting of potato seed tubers reduces apical injury to shoots and stem cankers. In practice, seed pieces are dropped in the usual trench with minimum covering, the soil being worked to the plants gradually in succeeding cultivations. Townsend (39) worked out a control for bottom drop of lettuce through the application of 20 to 25 lb. per acre of 2 per cent ethyl mercury phosphate dust under the nearly mature plants. The use of mercury compounds for control of brown patch of turf grasses is a common practice.

When seeds such as those of tomato, pepper, eggplant, and zinnia are infected, the usual chemical treatments are inadequate. A hot-water treatment is satisfactory for this purpose (2).

Treatment of potato seed tubers to rid them of the scab organism and the sclerotia of *R. solani* is an old practice (p. 152). Its value is limited by the extent of soil infestation. In the case of *R. solani*, a second limitation lies in the fact that, in the main, sclerotia borne on tubers are those of mildly pathogenic or saprophytic strains. In many states seed-tuber treatment for potatoes is no longer recommended generally. Small (35) reports no success with this method for control of *R. solani* on potatoes in England.

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WHITE HEART ROT OF DECIDUOUS TREES

There are numerous species of the Homobasidiomycetes which are associated with one or another decay of trees. One of the most common

and destructive heart rots is that known as white trunk rot, white spongy rot, or white heart rot. It is world-wide in distribution and occurs on many species including poplar, maple, oak, birch, beech, apple, and pear. It does not affect conifers.

Meinecke (8) found this disease the chief cause of cull logs in quaking aspen, with an average loss of about 18 per cent. There is an increase in

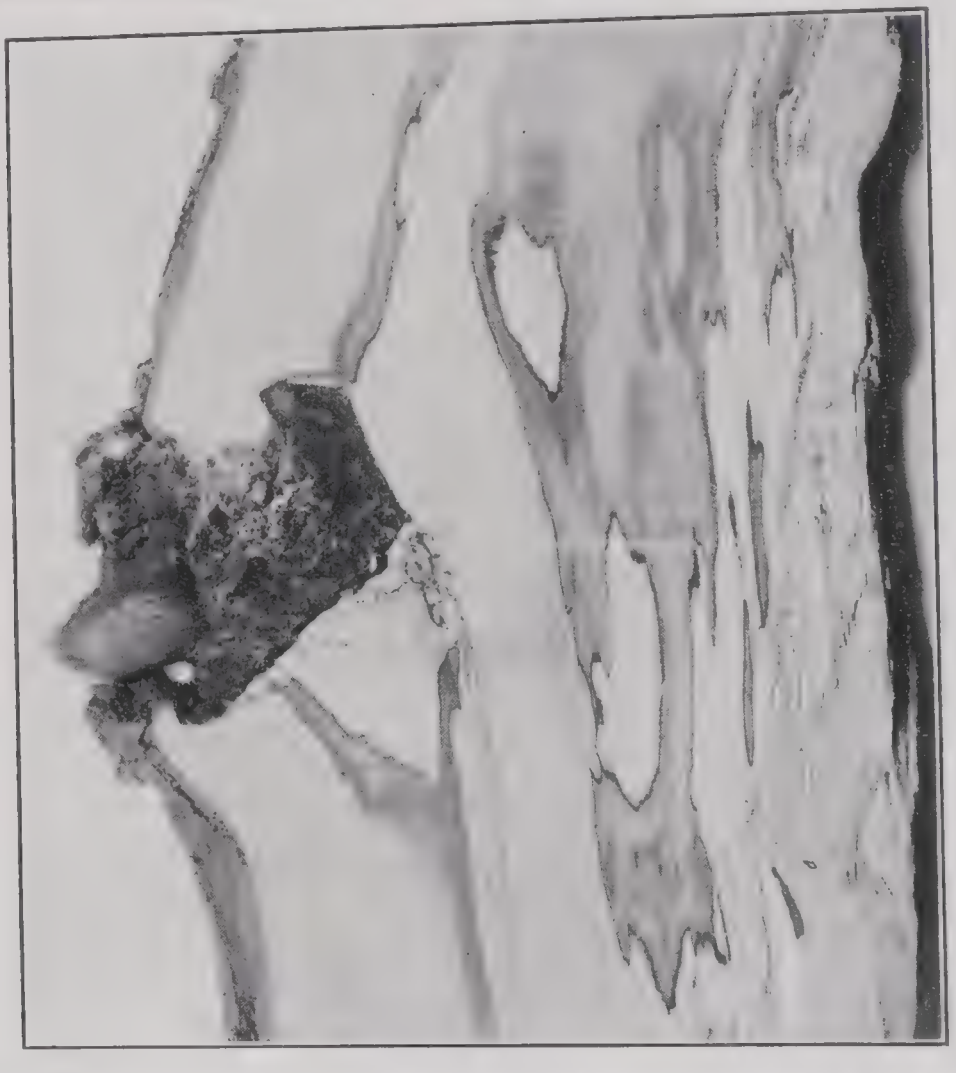

FIG. 146. White heart rot in hard maple with a sporophore forming in a knot. The zone lines are characteristic of the disease. (*Courtesy of U.S. Department of Agriculture.*)

percentage of gross cull with the age class of trees and a sudden increase as the trees approach 100 years of age. Schmitz and Jackson (10) in Minnesota found 4.8 per cent average loss at 30 years, 7.8 per cent at 40 years, 11.4 per cent at 50 years, 15.7 per cent at 60 years, and 20.5 per cent at 70 years. In many regions where aspen is important in paper production the losses sustained from white heart rot are of major significance. It is a common agency of decay in dead logs in the forest but is not important as an incitant of decay in sawed wood in storage yards or in constructed buildings.

Symptoms. The first evidence of the disease consists in yellowish-white areas in the heartwood surrounded by an invasion zone which varies in color with the host from yellowish green to brownish black (Fig. 146). The decayed wood gradually becomes soft and usually light in color with fine black lines traversing it. The heartwood of living trees is affected chiefly, but after such trees die, the dead sapwood is invaded. The fruiting bodies of the fungus, known as conks, appear as hard, woody, hoof-shaped "shelves" or "brackets" on the surface of affected logs, usually at knots or wounds (Fig. 147). They are dark in color on the upper surface, smooth and shiny at first, becoming rough, zonate, and cracked with age. The lower surface is brownish with numerous pores, the newly formed edge being yellowish brown at first. The interior is brownish and layered.

FIG. 147. White heart rot. Sporophores of *Fomes igniarius* on *Populus tremuloides*. (*Courtesy of U.S. Department of Agriculture.*

The Causal Organism. *Fomes igniarius* (Fr.) Gill., 1878.

SYNONYMY:

Boletus igniarius L., 1753.
Polyporus igniarius Fr., 1821.
Polyporus nigricans Fr., 1821.
Fomes nigricans (Fr.) Gill., 1878.
Phellinus igniarius Quel., 1886.
Mucronosporus igniarius Ell. & Ev., 1889.
Pyropolyporus igniarius Murr., 1903.

The fructification body (conk) takes the form of shelf-like perennial crusts or brackets on the tree trunk, varying from 1 to 12 in. in width. The lower surface bears the numerous pores within which basidia bear the basidiospores. A new layer of pores is produced each year over that of the preceding year. The conk may function for as long as 80 years. Tinder was formerly made from the conks; hence the name "tinder fungus" is often applied to the organism. Verrall (12) distinguished three types of the fungus, according to the host substrate, *i.e.*, aspen type, birch type, and miscellaneous-host type. Hirt (3) found that isolates from aspen, birch, beach, elm, and ironwood were all pathogenic on all of the hardwoods listed.

The pores are 100 to 149 μ in diameter; the dissepiments are 48 to 71

μ in thickness; setae are 11.9 to 19.0 by 4.6 to 6.9μ; basidiospores are hyaline, subglobose, 4 to 7 by 5 to 7.5 μ (9). Verrall (12) found that haploid lines from single basidiospores fall into two mating groups.

Disease Cycle. The basidiospores produced in the perennial conks are discharged forcibly and carried by air currents. When spores germinate on the surface of the tree, the mycelium invades through wounds, principally in old branch stubs. Invasion of the heartwood follows, the organism penetrating the cell walls and producing in the latter holes of relatively large bore. The young hyphae are hyaline, but older mycelium is brownish. It is abundant in old, white, decayed wood, relatively sparse in the yellowish-green or dark outer zone. It is absent in nondiscolored wood of maple, and in aspen it may extend up to $^3/_4$ in. in a radial direction and up to $2^1/_{16}$ inches in a longitudinal direction beyond the line of distinct discoloration (6). In the early stages of the disease the hyphae are most abundant in the medullary rays, and from them they pass into the wood cells through the pits. As the wall around the pit is dissolved, the latter enlarges. The fungus is capable of disintegrating the lignin of the cell wall. As the decay advances, the hyphae become more abundant, binding the disintegrated material into a loosely compacted mass. Advance of the fungus in heartwood may lead to encroachment upon the sapwood. When the new sapwood becomes involved, dead areas in the bark appear, and if the entire diameter of the new sapwood is concerned, the tree is soon killed. The fungus continues to grow in dead trees, sometimes at an accelerated rate with production of more conks. The fungus has an optimum temperature of 24 to 27°C. for growth on malt agar (12). The disease occurs in relatively humid and very dry regions, in tropical and in temperate zones.

Control. The only feasible means of reduced damage in the forest must start with management of relatively young stands. Infected and dead trees should be removed, and all practicable measures taken which tend to reduce injuries. In kiln-dried wood the fungus is killed. Handling of aspen logs for paper pulp should take into account the fact that the fungus continues to produce decay in the felled log and that moist environment tends to speed the process. In view of the fact that the fungus invades chiefly through old branch stubs, valuable shade trees should have all wounds produced by trimming or by other means treated with creosote-tar paint or some other suitable fungicide.

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ARMILLARIA ROOT ROT

While most of the agarics, or gill fungi, are saprophytes, one species, *Armillaria mellea* (Vahl) Quel., is a widespread incitant of root rot of forest and orchard trees. It is pathogenic on many other plants; *e.g.*, it incites a dry rot of potato tubers. The disease was first connected with the fungus in 1873 by Hartig, who continued to study it on forest and fruit trees during the following 30 years. Much of the earlier work is reviewed by Thomas (9).

In the United States the disease is probably most destructive to stone-fruit and *Citrus* trees in the Pacific coast states. Benton and Ehrlich (2) in a survey in northern Idaho found *A. mellea* the most common local associate of root disease in western white pine, *Pinus monticola* Dougl.

Symptoms. The disease is initiated by invasion of the root system of the tree. After the pathogen makes sufficient progress, various nondescript manifestations of unthriftiness follow, but nothing characteristic is to be noted until the fruiting bodies of the fungus appear, usually in large numbers around the base of the trunk (Fig. 148). They may be seen also around the stumps of dead trees which have been removed. The fruiting body is a mushroom, and the honey-colored top (**pileus**) is speckled and has a viscid appearance. On the lower side of the pileus are white **lamellae,** or **gills,** while slightly below the pileus on the **stalk,** known as the **stipe,** are the remains of the inner veil of the immature fruiting body, known as the **annulus.** The stipe is somewhat swollen at the base. Another conspicuous form of the fungus which helps to distinguish the disease consists of hard black strands which ramify through the soil and along roots and

trunks. They are the **rhizomorphs** of the fungus, often referred to popularly as "shoestrings."

The fruiting bodies develop at the ends of the rhizomorphs. As the cambium of the tree is invaded, the top gradually withers and dries out, and the tree eventually dies. When potatoes are grown on soil where rhizomorphs are present, the latter adhere to the skin of the tuber and permeate it at various points. Shallow corky areas, which occasionally extend to the pith, consist of light brown tissue interspersed with convoluted, white, brown-edged, mycelial plates.

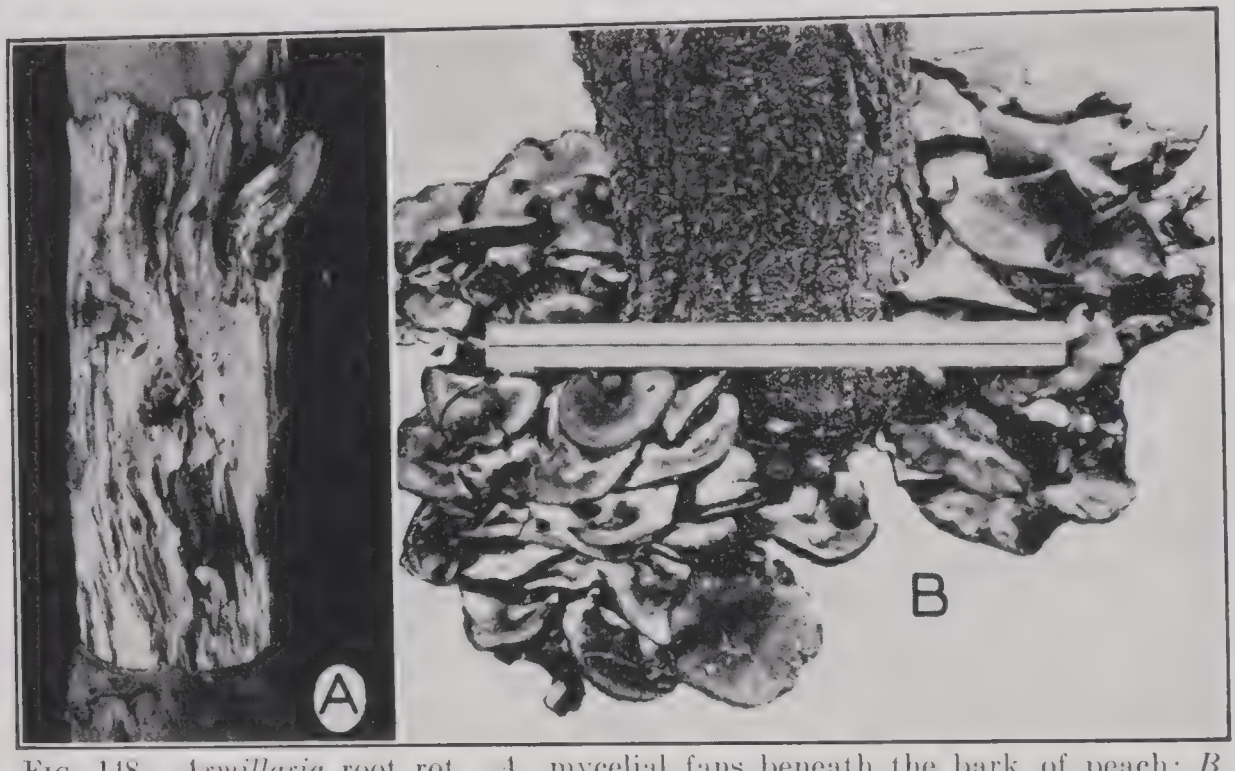

FIG. 148. *Armillaria* root rot. *A*, mycelial fans beneath the bark of peach; *B*, sporophores at the base of an apricot tree. (*Courtesy of M. W. Gardner.*)

The Causal Organism. *Armillaria mellea* (Vahl) Quel.

SYNONYMY: *Agaricus melleus* Vahl, 1777.

The mycelium develops in white, fan-shaped felts between bark and wood, and the felts are later replaced by subcortical rhizomorphs, which enter the host chiefly through medullary rays. The superficial rhizomorphs are string-like with an apical growing region. The development of the fruiting body is described by Atkinson (1). In northern regions they usually appear in the autumn and may not occur until the tree is dead or nearly so. The stipe is up to 10 in. long; the top of the pileus is honey-colored with scales; the gills are white, becoming darker with age. The stipe is usually dark-colored and has an annulus. The hymenium bears

basidia, interspersed with paraphyses, each bearing hyaline, elliptical or slightly reniform spores (6 by 9 μ) on sterigmata (Fig. 149). The spores are discharged in great numbers and are air-borne.

Disease Cycle. The fungus subsists on roots and stumps in the form of rhizomorphs. The latter become attached to the root, in part at least, by the hardening of a resinous substance which envelops the rhizomorph near its white growing tip. Hyphae penetrate the outer cells of the root as an anchor. Branches of the rhizomorph arise from the side in contact with the root. In fleshy roots or tubers, as well as in woody roots, penetration occurs by the rhizomorph branch acting en masse rather than by individual hyphae. The initial invasion appears to be by mechanical force. Later,

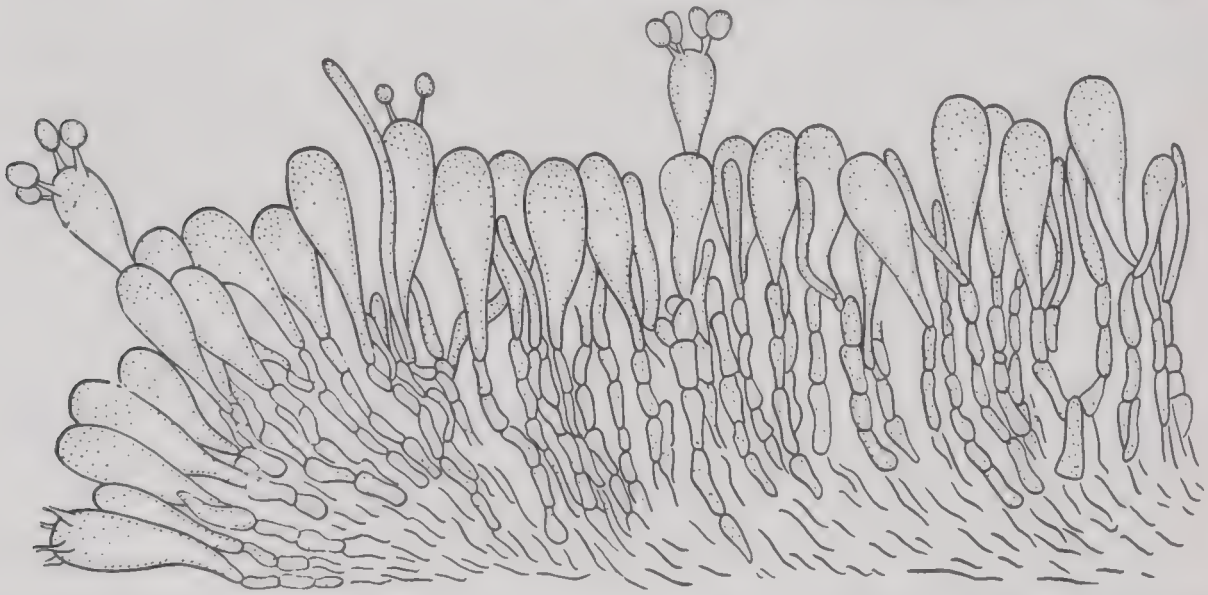

FIG. 149. *Armillaria mellea*. Sporiferous layer showing basidia, sterigmata, and basidiospores. (*After Hartig.*)

secondary branches and single hyphae penetrate the fleshy tissues and break down the cells by enzymic as well as by mechanical action. In relatively resistant roots, such as those of pear, the fungus penetrates in the usual manner but fails to establish itself, and the host lays down cork to wall off the wound produced by the invading fungus (9). The basidiospores are not regarded as important in infection of the living host; instead they initiate saprogenic development on dead stumps, from which rhizomorphs are formed (7).

Benton and Ehrlich (2) found the optimum temperature for growth of the fungus on malt agar to be 21 to 25°C. Bliss (3) studied the development of root rot on *Citrus* and *Prunus* seedlings in soil kept at constant soil temperatures. *Citrus* plants (sweet and sour orange) showed most severe disease at 10 to 18°; the greatest root growth was at 17° and 31°. In peach and apricot the disease was most severe at 15° and 25°; roots grew

most rapidly at 10° to 17°. It appears that the fungus was most pathogenic at temperatures at which host growth was slow and least so at the temperatures which promoted most root development. In other words the influence of temperature on the host had more effect on host-parasite interaction than the influence of temperature on the organism. Childs and Zeller (5) studied the disease on orchard trees in Oregon planted on fir-cleared land and oak-cleared land. They found evidence of two races, the oak race being parasitic and the fir race apparently not so.

Control. This disease is a difficult one to control on woody plants in regions where soil and climate seem to combine to favor its development. One of the serious problems is in Pacific coast stone-fruit orchards, where occasional trees become infected and die. Local sterilization of the soil is necessary before such trees can be replaced. A common precaution is to trench the infested area occupied by the tree to prevent growth of rhizomorphs through the soil to infect roots of healthy trees. This precaution has had only limited success in some areas (10).

Fleshy-root and tuber crops should not be planted in recently reclaimed forest land where *Armillaria* has been prevalent. Several years' cultivation with nonsusceptible crops should intervene until roots and other debris harboring rhizomorphs have disappeared.

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CHAPTER 10

DISEASES INCITED BY PHANEROGAMIC PARASITES

Relatively few diseases of higher plants are induced by phanerogamic parasites. In tropical regions many epiphytes (p. 8) occur which are nonparasitic and in the main do not incite disease development except by indirect means. The Spanish moss, *Tillandsia usneoides* L., belonging to the pineapple family (Bromeliaceae), which grows profusely on live oak and southern cypress, may at times kill trees by reduction of light. Climbing vines such as bittersweet (*Celastrus scandens L.*) may injure hardwood trees or kill them by strangulation. Among the most strictly parasitic forms are the true and dwarf mistletoes, belonging to the Loranthaceae, and the dodders, belonging to the Convolvulaceae.

THE TRUE MISTLETOES

In Europe *Viscum album* L. is the most widespread species on hardwoods and conifers. Other species more restricted in occurrence are *V. cruciatum* Sieb. on hardwoods and *Loranthus europaeus* Jacq. on oaks. In America the true mistletoes are all members of the genus *Phoradendron*, which in turn is restricted to this hemisphere. They are not found in northern states having severe winters. The approximate northern limits are Oregon on the Pacific coast, southern Colorado and the Ohio River in the Middle West, and southern New Jersey in the eastern states. They extend southward into Mexico, Central America, West Indies, and South America. There are some 70 distinct species of *Phoradendron* according to the comprehensive monograph by Trelease (7). Many hardwoods and conifers are affected. Various degrees of damage to the value of the forest tree occur, and in some instances trees are killed.

Symptoms. The conspicuous sign of the disease is the presence of the parasite. The portion of branch affected is atrophied owing to stimulation of excessive growth and abnormal swellings. The branch usually dies beyond the point of infection. In some hosts, such as osage orange, witches'-brooms are produced.

The Causal Organisms. Among the more common species are the following: The eastern mistletoe (*Phoradendron flavescens* Nutt.) with white berries extends from New Jersey to Missouri and south to the Gulf of Mexico. It occurs on oak, elm, maple, sycamore, gums, and other hardwoods. The Texas mistletoe (*P. engelmanni* Trel.), also with white berries, occurs on oak, elm, mesquite, osage orange, and hackberry. Arizona mistletoe (*P. macrophyllum* Cockerell), with white berries, attacks alder, ash, poplar, hackberry, sycamore, walnut, and willow. California mistletoe (*P. cali-*

fornicum Nutt.) is leafless, has long stems and reddish-pink berries, and occurs chiefly on mesquite and other leguminous trees. Juniper mistletoe (*P. juniperinum* Engelm.), from Colorado to Mexico, and constricted mistletoe (*P. ligatum* Trel.), from Oregon to Mexico, are leafless species restricted to junipers. The incense-cedar mistletoe (*P. libocedri* Howell) is restricted to the incense cedar in Oregon, California, and Nevada.

The plants are leathery with yellow to dark green leaves, or without leaves, growing upright from the point of attachment. The plants are dioecious, the pistillate plant bearing groups of white, yellow, or pink berries, depending upon the species. The berries contain mucilaginous material around the enclosed seeds by means of which the latter are attached to the host or to birds which disseminate them.

Disease Cycle. The sticky coating on the seed is the means whereby the latter becomes attached to the bark of the tree. After the seed germinates, the shoot coming in contact with the bark flattens out to form an attachment organ from which a papilla-like primary haustorium penetrates the bark through lenticels or axillary buds. Only relatively young branches are invaded. The branches of the haustorium ramify through the living bark. Other branches advance radially into the cambium. These radial branches, known as sinkers, remain active and become more deeply embedded in the wood as successive annual rings form. The mistletoe plant, once established, may live as long as the tree. Wagener (9) reports a case of *P. libocedri* on incense cedar 448 years old in which the parasite had lived for at least 409 years.

The evergreen leafy or leafless portion of the parasite consists of shoots which arise from the attachment disks. Stems are round and jointed, growing relatively slowly. Leaves and stems contain chlorophyll, water and minerals being supplied by the host. The plants require ample sunlight for development and in thick forests are found to be most prevalent in the upper parts of tall trees.

Control. In shade and ornamental trees young branches should be cut a foot or more below the point of infection. In older branches the bark and wood must be cut out for a foot or more away from each infection, and disinfectant should be applied to the wounded surface. In managed forest stands infected trees should be removed as soon as practicable.

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THE WESTERN-YELLOW-PINE MISTLETOE

This is a destructive disease of the western yellow pine (*Pinus ponderosa* Laws.) and related species. The parasites are dwarf mistletoes belonging to the genus *Razoumofskya* (*Arceuthobium*).

Symptoms. The first sign of infection on a limb is a fusiform swelling. Such swellings may occur on the bole of the tree, where they may result in burls if the tree is infected while young. When old boles are infected, the swelling may be quite inconspicuous. The hypertrophy is often accompanied by a resin flow appearing as small drops on the lower sides of heavily infected limbs. The parasite forms aerial shoots, which die after flowering and fruiting and fall from the tree (Fig. 150).

The Causal Organisms. *Razoumofskya cryptoboda* (Eng.) Cov. and *R. campylopoda* (Eng.) Piper. Seeds are borne singly in berries, where they are embedded in mucilaginous pulp. The berries are borne on short pedicels, and at maturity the seeds are ejected forcibly for several yards. Birds and animals aid in wider distribution. The sticky seeds become attached to the tree, germinate, and develop holdfasts. Penetrating haustoria invade young bark, producing haustoria and sinkers. Aerial shoots arise from the haustorium, bearing flowers after 2 years or more. Plants are dioecious. After flowering the male plants die, and female plants die after fruits are mature. New shoots arise, however, from the cortical haustorium.

Control. The elimination of infected trees from managed stands is the only means of control.

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DODDER

There are over 50 species of *Cuscuta* found in North America; most of these are native. Some of the introduced species are the most troublesome. Clovers, alfalfa, and lespedeza are the most seriously affected crops,

FIG. 150. Dwarf mistletoe, *Razoumofskya campylopoda*, on *Pinus jeffreyi*. (*Courtesy of U.S. Department of Agriculture.*)

but dodders may also become important on sugar beets, onion, and flax. They also occur on many other crop plants, ornamentals, and wild species. The cereals are not affected.

Symptoms. The parasite first becomes noticeable as a slender vine which, as it grows, entwines its yellowish, almost leafless stem around the

above-ground parts of its host (Fig. 151). The color varies from almost white to yellow tinged with red or purple. The leaves are reduced to very small scales. The parasite later produces inconspicuous flowers (white, pink, or yellow, depending upon the species). Dodder appears in patches of various sizes in a field. In perennial crops such areas increase in size in successive years. As host plants are infected, they decline in vigor as the dodder thrives. Infested areas gradually take on the yellowish color of the parasitic vine.

FIG. 151. Dodder (*Cuscuta* spp.) on onion.

The Causal Organisms. *Cuscuta* spp. The species are not readily distinguished except by study in the laboratory. Some species have a relatively wide host range, *e.g.*, common dodder (*C. gronovii* Willd.) and field dodder (*C. pentagona* Engelm.), while others show distinct preferences for certain crops. Small-seeded alfalfa dodder (*C. planiflora* Ten.) and large-seeded alfalfa dodder (*C. indecora* Choisy) are seldom harmful on crops other than alfalfa. The flax dodder (*C. epilinum* Weihe) is confined chiefly to the flax crop.

Dodder seeds are grey to reddish brown and in most species are smaller than those of red clover and alfalfa.

Disease Cycle. The dodder plant reproduces from seeds which over-winter in the soil or as contaminants in crop seeds. Dodder seeds may remain viable for several years. The seed germinates in the soil, the plant forming a very fragmentary root system and sending up a yellowish shoot which makes contact with the nearest green plant. If such contact is with a suitable host, the vine climbs in an encircling manner. The dodder may subsist temporarily on an uncongenial host and later reach a congenial plant, on which its development continues almost exclusively. If no host is available, the dodder vine lies on the ground and dies within 4 or 5 weeks.

Once established on a congenial host, and having used up the nutrient reserves in the seed, the dodder promptly sends haustoria into the stem or leaf; these penetrate to the fibrovascular system. The haustoria, commonly called suckers, excrete enzymes such as diastase which hydrolyze the starches and other potential food materials in the host, and they serve as the channel whereby the parasite receives nutrients during the rest of the

season. Pieces of the established vine may be broken off, and when dropped on uninfected plants, they can establish themselves and start new centers. The dodder vine extends from plant to plant, building up a tangled mass as the season progresses and gradually suppressing host development. After midseason, flowers form, and seeds are produced. Some species produce very little or no seed in certain locations. For example, the clover dodder (*C. epithymum* Murr.) rarely goes to seed in the United States, but it may under special circumstances live over by means of stems.

The dodder is distributed over long distances with crop seeds. It is disseminated locally in freshly cut hay, by moving about of man and animals, by farm implements, by irrigation and surface drainage water, and by fresh manure from animals fed forage containing the seeds.

Control. Dodder can sometimes, but not always, be removed from crop seeds by the use of proper cleaning machinery. The removal of seeds of the large-seeded alfalfa dodder is practically impossible. Clean seed is, of course, the first requisite of control. When dodder gains a foothold, certain seasonal measures are important. In order to prevent maturing of seed, patches should be located early and the crop mowed before seeds are produced. If seeds have already been formed, the mowed plants should be raked, sprinkled with oil, and ignited when thoroughly dry. The seeds remaining on the soil can be destroyed by covering with straw and burning or by applying a blowtorch to the area. When large areas are infested, special procedures of cutting and feeding the hay must be followed (1).

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CHAPTER 11

DISEASES INCITED BY NEMATODES

The nematodes are roundworms which live in soil or water. Many are free-living; others are parasitic on animals or plants. Among the animal parasites are *Trichinella spiralis*, incitant of trichinosis in man, and *Necator americanus*, incitant of the hookworm disease in man. Most of the species of importance as plant pathogens are within the family Anguillulinidae, belonging to the class Nematoda and phylum Nemathelminthes.

The elongate worm is covered with an impermeable, smooth or transversely striated cuticle, beneath which is a subcuticular layer, and beneath the latter is a muscular layer. The body usually tapers at each end. At one end is the mouth, provided with papillae, leading to a buccal cavity which in turn leads to the esophagus. A valve is located at the junction of esophagus and intestine, the latter opening into the rectum and anus at the posterior end of the body. In the body cavity between wall and organs is a colorless liquid. Early in the development of the worms they differentiate into male or female forms. Further anatomical details may be found in the monograph by Goodey. The two genera of plant-parasitic nematodes considered herein are distinguished as follows:

Ditylenchus (*Anguillulina*) (*Tylenchus*). Adults of both sexes elongate; in female, vulva equatorial with paired gonads or postequatorial with single gonad outstretched anteriorly; in male, tail region tapering, lateral caudal alae present, spicules paired, gubernaculum present, pair of postanal papillae sometimes present.

Heterodera. Females swollen and sac-like, vulva terminal or subterminal, gonads paired. Males worm-like, tail bluntly rounded, lateral caudal alae absent, spicules paired, gubernaculum present, caudal papillae minute.

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ROOT KNOT

Root-knot larvae were first recorded in 1855 by Berkeley who found them in roots of infected cucumbers growing in a greenhouse in England. The disease affects a very extensive list of host plants (3, 4). It is most common on outdoor plants in tropical and subtropical climates and is relatively sparse in areas with severe winters except in greenhouses. In culture under glass it is a major disease where soil is not treated annually. In favorable climates crops growing on sandy soils are usually more subject to root knot than are those on heavy soils.

Symptoms. The root-knot disease is very distinctive because of the galls produced on roots and tubers. The above-ground symptoms consist chiefly in foliage color paler than normal, unthrifty development, dwarfing, wilt in hot dry weather, and sometimes death. Large and small roots contain swellings which vary from spheroid galls to elongate spindles (Fig. 152). The hypertrophy usually involves the entire root in the zone concerned

FIG. 152. Nematode galls incited by *Heterodera marioni*. *A*, on tomato; *B*, *C*, on parsnip. (*After Cox and Jeffers.*)

rather than taking the form of easily detached galls like those produced by nitrogen-fixing bacteria on roots of legumes. When young potato tubers are invaded, the parasite occupies a zone just beneath the skin, and the outer surface assumes a roughened warty appearance.

The Causal Organism. *Heterodera marioni* (Cornu) Goodey, 1932.

SYNONYMY:

Anguillula marioni Cornu, 1879.
Heterodera radicicola (Greef) Müller, 1884.

Heterodera javanica Treub, 1885.
Anguillula arenaria Neal, 1889.
Tylenchus arenarius Cobb, 1890.
Meloidogyne exigua Goeldi, 1892.
Anguillula vialae Lavergne, 1901.
Oxyuris incognita Koford & White, 1919.
Caconema radicicola (Greef) Cobb, 1924.

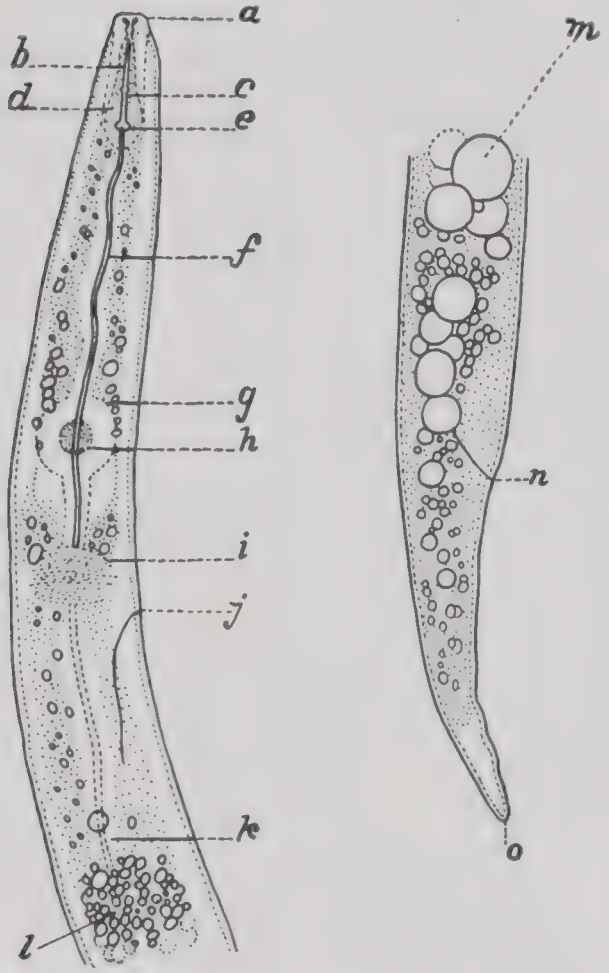

FIG. 153. Larva of *Heterodera marioni*: *a*, anterior end; *b*, *c*, *e*, stylet; *d*, buccal cavity; *f*, esophagus; *g*, *h*, outer and inner portions, respectively, of esophagal bulb; *i*, nerve ring; *j*, excretory port; *k*, *l*, lumen and thick wall, respectively, of alimentary canal; *m*, possibly a fat globule; *n*, anus; *o*, posterior extremity. (*After Bessey; original drawing by W. F. Chambers.*)

The eggs (30 to 52 by 67 to 128 μ) are ellipsoidal, sometimes slightly concave on one side. Larvae in the first stage are 12 to 15 by 375 to 500 μ (Fig. 153). The worm-like male attains a size of 0.03 to 0.36 by 1.2 to 1.5 mm.; stylet, 18 to 20 μ; spicules, 35 μ; gubernaculum, 10 to 12 μ. The female is pear-shaped, 0.27 to 0.75 by 0.40 to 1.30 mm., with a neck 0.15 to 0.24 mm. wide at the base and stylet, 10 to 12 μ. After fertilization, eggs are laid in a gelatinous mass extruded from the vulva, unless egg laying is impeded in the host tissue, in which case they may be retained within the body. From 300 to 600 eggs are laid by a female. There may be parthenogenetic reproduction in heavily parasitized roots. After larvae penetrate roots, the sexes are indistinguishable up to 15 days (Fig. 154). In the male a new cuticle is laid down inside the old one, and the male is produced within, coiled upon itself. In the female the new cuticle is laid down; the contents begin to swell; and organs begin to form. The larva first penetrates the meristematic tissue and migrates until the head becomes established in an intercellular space near the endodermis. The females remain in this position permanently, and the males do so until the final molt. The head of the nematode moves back and forth in the space as its lips make contact with the cells. As the parasite grows, the host cells become hypertrophied, and the former

becomes more closely appressed by adjacent walls. The stylet tip passes through the wall, and pulsation begins. The stylet is withdrawn within a few minutes to an hour or more, and another cell is penetrated. In this manner the parasite feeds within the cell without killing it, and it in

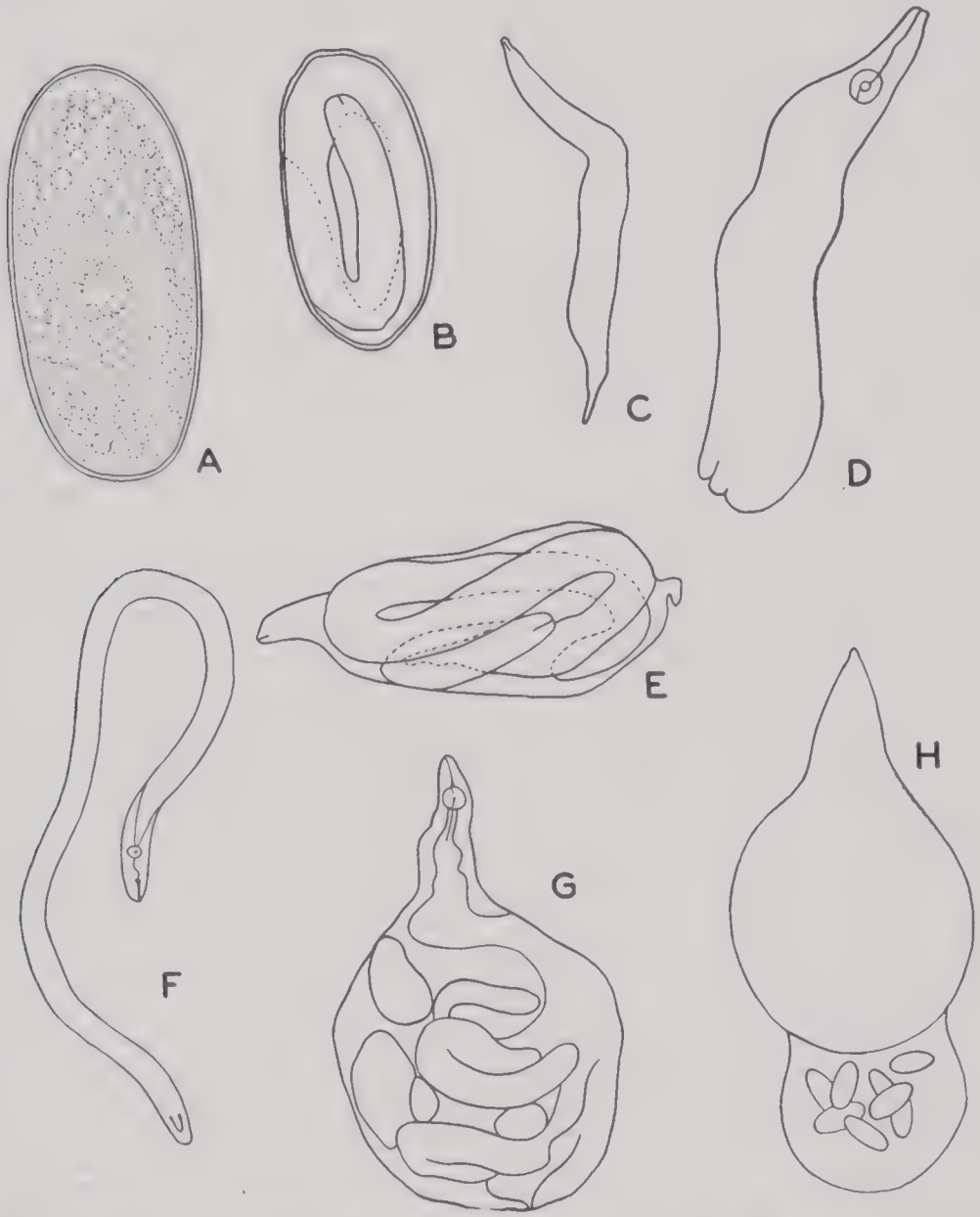

FIG. 154. Stages in life cycle of *Heterodera marioni*. *A*, egg; *B*, larva developing within egg; *C*, young larva before sexual differentiation is apparent; *D*, sexually mature female nematode; *E*, mature male still within larval skin; *F*, mature male; *G*, egg-bearing female nematode; *H*, female which has deposited eggs. (*A after Cobb; B–G, after Bessey; H after Christie.*)

some way stimulates host cells to enlarge and divide, leading to the characteristic gall formation (17).

The species contains a number of races distinguished by the hosts which they prefer (20).

Disease Cycle. The nemas live from one crop season to the next in the soil. Ordinarily, northern soils which freeze to a considerable depth in winter months are unfavorable to survival, but differences between races in respect to freezing injury may exist. Survival is common, for instance, on sandy soils of Long Island. The chief means of overwintering in the North is in greenhouse soils. Movement in the soil is slow. A larva may travel about 1 ft. per month. Larvae are found as deep as 27 in. and occasionally on tree roots in sandy soil as deep as 8 ft. The bulk of the population, however, is in the upper 2 ft. of soil. Since larvae and eggs are killed at temperatures between 40 and 50°C., there is probably much reduction in population in the upper 2 or 3 in. of soil in hot summer weather. A young free-living larva develops into an egg-laying female in about 80 days at 14° and in 16 days at 27°; development is retarded above 27°. Roots are infected over a range of 12 to 35°. Little disease develops below 16°, and cool-temperature crops such as lettuce can be grown with little damage in greenhouses held at 13° or below. On tobacco, galls are larger but less numerous at 19° than at 25°, and the effect upon the host is more pronounced at the latter temperature.

Larvae are transported long distances in planting stocks of various types.

Control. In greenhouses, hotbeds, cold frames, and other intensive cultures soil treatment is economical and is the chief means of control (p. 661). In many agricultural areas where mild winters permit survival the problem of control in field cultures has long been studied, and to a large extent practical means of effective control are still lacking. Various methods of destroying or reducing the nema population have been used with varying success. Those which have application in one situation or another have been summarized and evaluated by Kincaid (16). They include green-manuring crops, flooding periods, fallow periods, fertilization, and rotation with nonsusceptible crops. In the case of crops of high value, such as pineapple, the use of soil fumigants has met with considerable success.

While tolerant and resistant strains of some crops are known and, in so far as they are adaptable, are useful in reducing damage, this approach in control of root knot has not reached a point where it is used very generally. The extensive list by Tyler (22) records the observations on resistance and susceptibility of many species and varieties. Smith (19) made an extensive survey of nematode resistance in wilt-resistant cottons. He found that varieties developed for wilt resistance in the sandy soils of southeastern United States, where nematodes are prevalent, showed appreciable resistance to root knot. Those developed for wilt resistance in the heavy soils of Mississippi and Louisiana, where there are few root-knot nematodes showed little or no resistance to root knot. This seems to indicate that re-

sistance to root knot could be improved by intensive breeding with that objective.

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GOLDEN-NEMATODE DISEASE OF POTATO

This disease is commonly referred to as "potato sickness" in the British Isles, where it has been serious for many years, being first recognized in Yorkshire in 1903. It is prevalent throughout most of the northern potato-growing regions in Europe. The disease was discovered in 1941 in Long Island, where it had probably been increasing in severity slowly since about 1930. Up to 1949 it was confined to a relatively small area.

Potato and tomato are the chief economic hosts, but many species of the Solanaceae are susceptible, as well as lamb's-quarters, *Chenopodium album* L.

Symptoms. The signs of the disease are very indistinct. Delay in emergence, stunting, unthriftiness, and production of small tubers are the main effects. Detection of newly infested soil areas is difficult until the organism has become quite plentiful. The detection of female cysts attached to invaded roots is the only means of final diagnosis.

The Causal Organism. *Heterodera rostochiensis* Wr., 1923. The organism was regarded as *Heterodera schachtii* Schmidt until elevated to a distinct species by Wollenweber in 1923. Zimmerman in Germany designated it as *H. schachtii* f. *solani*. The form on beet is known in the western United States. Wollenweber points out that in contrast with *H. schachtii* the mature females of *H. rostochiensis* are almost spherical.

Disease Cycle. Eggs and larvae overwinter in the body of the dead female, which has been transformed into a brown, thick-walled cyst. In the spring and continuously throughout the season eggs hatch, and larvae emigrate to attack roots of susceptible plants. The larvae penetrate root or tuber, taking a position with head near the vascular system and the posterior part of the body often exposed. The first two molts occur within 5 to 7 days. About the tenth day the posterior part of the body enlarges as the third molt occurs, and during the next molt, which follows promptly, the female posterior enlarges rapidly to form a spheroid shape, while the male continues a vermiform shape. The spheroid female develops a cuticle and turns opaque, waxy, or pearly white. It now becomes macroscopic, turns yellow, produces eggs, and, as the season progresses, becomes golden in color, finally developing into a brown overwintering cyst. The cysts are known to remain viable in the soil up to 8 years.

Control. When the nematode was located in Long Island, quarantine measures were applied. Implements moved out of the area were treated, and potatoes were restricted in movement to markets where they would be used only for table purposes. No satisfactory control has been worked out

in Europe other than long rotation. Development of effective chemicals for soil treatment offers the most hopeful possibility.

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THE STEM- OR BULB-NEMATODE DISEASE

The parasite concerned is sometimes called the teasel nematode because it was first described by Kühn in 1859 in the flower heads of teasel, *Dipsacus fullonum* L. During the next 50 years similar forms were found in a number of crop plants, and some of them were given distinct specific names. It received a great deal of attention when it became prevalent on hyacinth and narcissus in Europe and on this account became the basis of a quarantine of the bulbs of these crops by the United States authorities. It is now world-wide in occurrence, and as with the root-knot nematode a number of physiologic races occur, and an extensive list of hosts is known (12). The potato-rot nematode, once regarded as a member of the species, has been given specific rank by Thorne (13).

Symptoms. In oats and rye, swelling, twisting, and waviness of leaves and stems, excessive tillering, loss of normal green color, and stunting are the major signs. Larvae may occur between swollen aborted glumes of oat. In red clover and alfalfa, short, swollen, stunted stems with numerous short side stems give a tufted appearance to affected plants. Young onion bulbs become swollen and misshapen. Hypertrophy and hyperplasia occur, and bulbs become soft. In hyacinth pustular areas occur on the leaves; flower stalks are stunted, and flowers are malformed; leaves eventually become twisted and dwarfed. The bulb scales become thickened by hypertrophy of the cells and turn brown. In narcissus small blister-like swellings appear on the leaves; in these the epidermis splits, and the tissue turns brown. Larvae enter the necks of the bulbs and invade the scale tissues including the flower rudiments, making bulbs flowerless the next season. The larvae migrate through the bulb tissue during storage.

The Causal Organism. *Ditylenchus dipsaci* (Kühn) Filipjev, 1936.

Synonymy:

Anguillula dipsaci Kühn, 1857.
Anguillulina dipsaci (Kühn) Gerv. & V. Ben., 1859.

Anguillula devastatrix Kühn, 1868.
Anguillula secalis Nitschke, 1868.
Tylenchus askenasyi Bütschli, 1873.
Anguillula putrefaciens Kühn, 1877.
Tylenchus havensteinii Kühn, 1881.
Tylenchus hyacinthi Prillieux, 1881.
Tylenchus allii Beijerinck, 1883.
Tylenchus devastatrix Ritz-Bos, 1888.
Tylenchus dipsaci (Kühn) Bastian, 1865.

The eggs are variable in size and shape. Larvae when newly hatched are about 11 by 380 μ. Dimensions of adults are as follows:

Female: 0.04 to 0.06 by 1 to 1.8 mm.; esophagus, 0.16 to 0.24 mm.; tail, 0.08 to 0.12 mm.; stylet, 11 to 13μ.
Male; 0.03 to 0.04 by 0.9 to 1.6 mm.; esophagus, 0.16 to 0.22 mm.; tail, 0.08 to 0.1 mm.; spicules, 0.025 to 0.028 mm.; gubernaculum, 0.01 to 0.012 mm.

There are numerous physiologic races of the parasite (7, 8).

Disease Cycle. The larvae and eggs withstand freezing. They are sensitive to high temperatures, making disinfection of bulbs by hot water possible at 43 to 44°C. for 3 hr. The larvae remain viable in dry hay or bulbs up to 6 years. The parasite is distributed in bulbs, hay, and seeds. It is disseminated locally in irrigation water, by implements, and by vehicles, animals, and man. The larvae invade stomata, lenticels, around emerging rootlets. The larvae are intercellular, the middle lamellae being digested. The cells tend to enlarge and become more spherical, inincreasing the volume of intercellular spaces and giving the tissues a glistening appearance In leaf tissue invasion is chiefly in the spongy parenchyma.

Control. In bulbs of narcissus and hyacinth a hot-water treatment at 43 to 44°C. (110°F.) for 3 hr. is the standard treatment (4, 10). In field crops long rotation is the only means of control.

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CHAPTER 12

VIRUS DISEASES

Virus diseases of plants exhibit a variety of symptoms covering as wide a range of host reactions as that encountered in the bacterial and fungus diseases. The most common evidence of disease expressed in the green tissues of higher plants is the alteration in the usual development of chlorophyll. Interspersion of various degrees of chlorosis with the normal green color of the leaf results in a mosaic pattern of yellow and green. This common symptom has given rise to the use of the term "mosaic diseases" to designate a large group of virus maladies. In another large group, referred to as the "yellows diseases" a nearly uniform reduction of chlorophyll is found with relatively little mottle or mosaic pattern. While these and other chlorophyll deficiencies have come to be regarded as common indicators of virus infection, it should be recognized that other causal agents, *e.g.*, certain mineral deficiencies, may bring about similar effects. Some of the other symptoms frequently encountered among the virus diseases are worthy of mention.

COMMON SYMPTOMS AND EFFECTS

Veinclearing and Veinbanding. There commonly appears in systemically infected leaves before mottle or mosaic a clearing or chlorosis of the tissue in or immediately adjacent to the veins. This pattern is referred to as veinclearing. Veinbanding consists of a broader band of chlorotic tissue along the veins, or it may consist of bands of green tissue in that position, set off by chlorosis or necrosis in the interveinal parenchyma. Veinclearing and veinbanding may be transient and precede mottle, or they may remain as the major symptom of the disease concerned, *e.g.*, potato veinbanding disease on tobacco.

Ring Spots. On inoculated or systemically infected leaves the symptoms appear in localized spots. These spots consist of various types of chlorosis and necrosis. They may be circular chlorotic areas, in which case they are called **chlorotic ring spots.** In other cases the necrosis may appear in rings alternating with normal green. Such spots are called **necrotic ring spots.** The centers of either type of spot may eventually also become necrotic.

Necrosis. In diseases of both the yellows and mosaic groups, necrosis appears in various forms. As mottle symptoms progress, the older chlorotic tissue may break down and necrotic spots appear. The necrosis may spread in extent and cause various patterns as it develops. In some cases necrosis is the predominant feature of the disease. It is likely to in-

volve parenchyma and veins of the leaf and the stems. In the latter case it is often referred to as **streak.** **Top necrosis** refers to relatively rapid killing of a bud, branch, or the entire top of the plant (Fig. 162). Some viruses do not infect a given host systemically but affect the tissue at points of inoculation by causing a localized breakdown. The size of such spots varies with the virus and host. Some are no larger than a pinhead; others are up to a centimeter in diameter. This type of reaction is known as local necrosis, and the spot is referred to as a **local lesion.**

Necrosis of the phloem elements appears at certain stages in some virus diseases. This was first described in connection with leaf roll of potato, where it occurs in the stem and tuber. The affected phloem tissue is particularly conspicuous in the tuber of certain varieties where a net-like necrosis is formed in the inner phloem. It occurs also in roots and leaves of sugar beets affected with curly top. It has been shown in the case of this disease that the virus moves through the plant primarily through the phloem. Various other manifestations of local necrosis may occur, *e.g.*, the breakdown of parenchymatous tissue in the pith of potato stems and tubers with the yellow-dwarf virus and in the interior of cabbage heads affected with the mosaic viruses.

Stunting and Premature Death. It is quite natural that the symptoms cited above would be associated with a stunting effect on the host plant. As a matter of fact virus diseases are, with few exceptions, hypoplastic in their general effect. This is shown in many cases by shorter internodes, smaller leaves and fruits, and reduction of size of various other parts. In vine crops the stunting is conspicuous as reduction in length of the internodes of the vines. In potatoes the tops are often reduced to a rosette. In fact virus-affected plants generally are more often stunted than killed. One outstanding exception to this is yellow dwarf of potato. This disease is favored by warm soil temperature when the seed tubers are sprouting. If the virus is present under such circumstances, death of the sprout commonly occurs before it reaches the surface of the soil or soon after that time, and the end result is a poor stand of the crop (Fig. 174). Premature dropping of lower leaves is sometimes a symptom of a virus disease. A good example of this is in cabbage mosaic, where sometimes premature defoliation may be the only conspicuous sign of the disease.

Malformations and Overgrowths. In many cases distortion of tissues is a characteristic symptom. Leaf roll of potato is so called because of the upward rolling of the margins of the leaflet parallel with the midrib. In some diseases a roughening of the leaf blade known as rugosity occurs, as in rugose mosaic of potato. Reduced development of the leaf blade in proportion to midrib leads to asparagus-fern-leaf symptoms in tomato mosaic. Increase in the number of leaflets leads to Boston-fern-leaf symptoms in the

same disease. In some ornamental plants affected with viruses, *e.g.*, sweet pea, petunia, stock, and tulip, the normal petal color development is interrupted, giving what is commonly referred to as broken flowers. The normal color is absent or is interspersed with abnormal shades in the same petal.

We find among virus diseases a wide range of abnormal growth reactions. Some of these are the result of retarded growth in a portion of the tissue of an organ. Thus cucumber plants affected with mosaic often produce distorted, warty fruits (Fig. 159). Some viruses incite formation of masses of hypertrophied tissue, known as **enations,** on the surface of leaf or stem. In other cases virus infection results in stimulation of dormant buds, hyperplastic tissue, and unusual differentiation. Spindle tuber of potato is an example of such distorted growth. Tubers of infected plants are more elongated than is normal and have correspondingly less breadth. More growing points or "eyes" are included in the enlargement of the stolon which becomes the tuber. The aster-yellows virus affects many species, and in most plants at some stage it causes proliferation of buds. Aster inflorescences are distorted because of excessive production of flowers; carrots are stimulated into excessive formation of top leaves and tufts of hyperplastic secondary roots on the fleshy taproot. An extreme example of atypical growth is found in the extensive stimulation of numbers of buds in the potato-witches'-broom disease. Even flowers are distorted so that small tubers are produced on flower pedicels while flower petals may emerge from the eye of such a tuber. Proliferation of stem buds extends to the underground stolons, where several hundred small tubers in a single hill may be found in place of a normal complement of six or eight of ordinary size. A virus has been described which causes distinct tumors or galls on roots and stems of a number of species of plants (20).

Masked Symptoms and Symptomless Carriers. There are many instances in which the virus is present in the infected plant but no distinguishable symptoms appear. This may be due to a particular set of environmental conditions under which the symptoms disappear. Under such conditions the plant is referred to as having masked symptoms. In mild mosaic of potato the characteristic mottle of the foliage is pronounced if the temperature is around 16°C., while if diseased plants are removed to a temperature of 24° for a few days, all visible signs of mottle disappear. If the same plants are returned to 16°, the mottle reappears.

In some diseases the virus infects a given host and increases therein, but no visible signs appear over the entire range of environmental conditions to which the host is usually exposed. This condition prevails in many varieties of potato infected with the latent-mosaic virus. It occurs in many varieties of plum infected with the virus of peach yellows or that of little

peach. In other cases a host may show signs of the disease for a relatively short time after it becomes infected, but the symptoms gradually disappear, and clones derived from such a plant continue to contain the virus but show no symptoms. Some potato varieties show symptoms of latent mosaic for a short time when first infected but eventually become and remain symptomless. Tobacco commonly outgrows the symptoms of the tobacco-ring-spot virus. Infected plants of this type are referred to as symptomless carriers. The plant which is a symptomless carrier is distinguished from one regarded as having masked symptoms by the fact that in the latter instance the signs are not visible in a particular sector of the usual range of environment, while in the former case the plant is symptomless throughout the entire range of environment.

TRANSMISSION OF PLANT VIRUSES

One of the cardinal characteristics of a virus is the fact that it is infectious. However, virus diseases differ from other infectious diseases in that the transmitted unit is invisible under the ordinary compound microscope. It was recognized early, however, that viruses differ widely in the means and in the ease with which they may be transmitted. This was brought about by the coincidence that the two virus diseases studied first, *i.e.*, peach yellows and tobacco mosaic, represent extremes in this regard. The common-tobacco-mosaic virus is transmitted readily by simple mechanical means. Such a procedure was of no avail with the peach-yellows virus, but its infectivity, by use of buds and grafts, was demonstrated.

It is natural that the method of transmission of the virus in question receives early attention in the study of a given disease. For the purpose of surveying this subject we may conveniently divide methods of transmission into the following categories: (*a*) mechanical means; (*b*) budding or grafting; (*c*) seed or other propagative organs; (*d*) insects; (*e*) parasitic phanerogams; (*f*) soil.

Mechanical Transmission. Transmission by this means consists in removal of the plant sap from a diseased plant by artificial or natural means and transfer to a healthy plant by rubbing or injecting. Since infection through natural openings, such as stomata, is rare, mechanical transmission practically always involves wounding of the inoculated plant to permit establishment of the infectious agent. The type and degree of wounding vary widely and in themselves have become one of the bases for characterization or diagnosis. For instance, the tobacco-mosaic virus is transmitted very readily by rubbing the extracted juice of a diseased plant over the leaf of a healthy tobacco plant, which process wounds hairs or epidermal cells sufficiently to bring about infection. Some viruses, because of a lower concentration of the virus, or because of the presence of an inhibitive sub-

stance, require more complete abrasion of the host tissue than others do to produce a high percentage of infected plants. This is commonly provided in experimental procedure by adding finely divided carborundum or other abrasives to the inoculum.

The readiness with which a virus is transmitted mechanically usually has a bearing upon the degree of natural spread. The tobacco-mosaic virus, for instance, is seldom spread by insects (in so far as is now known) but is transmitted chiefly by hands of workmen in the process of removing "suckers" in tobacco and in greenhouse-grown tomatoes. Cucumber mosaic is spread by pickers when the green fruits are removed and the hands or implements contaminated in the process. The spindle-tuber virus of potato is spread by the knife blade when seed tubers are being cut for planting.

As a class, the viruses in the mosaic group are most readily transmitted by mechanical means although there is a great deal of variation in the ease of transmission. Viruses of the yellows group, on the other hand, rarely lend themselves to transmission by this means.

Transmission by Grafting. Since viruses are intimately associated with the living cells of the host and since they are commonly distributed systemically throughout the living tissue, it is quite natural to expect that the transfer of the tissue of a diseased plant, by one or another means of grafting, to become an organic part of a healthy plant would be a satisfactory method of transmission. Thus most viruses which have been studied in this respect have been found to be transmitted by this method if satisfactory graft unions are secured. It is the most widely applicable means of transfer, but owing to the fact that it is cumbersome and time-consuming, it is not used for research purposes when simpler means are available. Furthermore, it becomes a means of natural transmission only in the case of plants in which grafting enters into normal propagation.

Many viruses were transmitted experimentally only by this means until an insect vector for the virus was known. Peach yellows is such an example. Some other virus diseases, *e.g.*, witches'-broom of potato, remain in the category of those in which grafting and transmission by dodder are the only known means of transfer.

Transmission by Seed and Other Propagative Organs. True seeds of higher plants are occasionally the means of virus transmission. Fortunately indeed, most viruses are not found in the mature seeds of their hosts. It has never been explained why the virus, occurring systemically in the host plant, is commonly found in the flower parts and in the immature seed but becomes entirely inactivated in the mature seed. The virus of common bean mosaic and that of southern bean mosaic are carried in the seed, but most other viruses affecting bean are not. The cucumber-mosaic

virus is transmitted occasionally in seeds of cucumber, muskmelon, and squash but commonly in the fleshy seeds of the wild cucumber, *Echinocystis lobata* Torr. & Gray.

Viruses are very commonly perpetuated in the vegetative organs of perennial plants. They are readily transferred from locality to locality in nursery stock, bulbs, tubers, and roots. Infected perennials are the common reservoir for overwintering of many viruses.

Insect Transmission (84). No other group of diseases is so generally dependent upon insects for dissemination of the infectious causal entity. Although most viruses are transmitted by insects, some very widespread and important ones are not as yet known to have insect vectors or are transmitted very infrequently by known vectors. One outstanding example is the tobacco-mosaic virus, which has not as yet been shown to be transmitted by insects from tobacco to tobacco although it has been transmitted, experimentally, from tomato to tobacco and tomato to tomato by aphids. Another example is the latent-mosaic virus of potato, which has as yet not been shown to be transmitted by insects. Both of these viruses are transmitted very readily by mechanical means. It is, of course, possible that insect vectors of these and other viruses in the same category will be discovered eventually.

Most vectors are sucking insects which belong chiefly to the Cicadellidae (leaf hoppers) and the Aphididae (aphids), while a few are members of the Aleyrodidae (white fly). The thrips (Thysanoptera), which are both sucking and rasping feeders, transmit a few viruses. Chewing insects are known to transmit viruses in relatively few cases.

It is of interest that practically all vectors of the so-called yellows group of viruses are leaf hoppers while practically all vectors of the mosaic group are aphids. In other words those viruses which are the more readily transmitted mechanically are aphid-borne and those which do not yield to such transfer are leaf hopper–borne. There are differences between vectors in their relation to the viruses they transmit. There is good evidence that some viruses transmitted by aphids are carried mechanically on the mouth parts of the insect, and the latter remains viruliferous for a period of only a few minutes to a few hours. The same species of aphid may transmit another virus differently in that it will not transmit the virus to a healthy plant until 6 hr. or more after it has fed upon the diseased plant. In such a case it is possible that the virus is ingested by the insect and is later transmitted through the body into the saliva, by which channel it eventually reaches the next host plant. The two groups of viruses are commonly referred to as nonpersistent and persistent. That the differences are those of the respective viruses is shown by the fact that one virus may be persistent and another virus nonpersistent in the same strain of aphid. It should also

be emphasized that insects which are not vectors of a given virus may take it up from the host and ingest it without transmitting it, and later discharge it in the feces. A given species of aphid may transmit several distinct viruses and not transmit others. Some viruses are transmitted by as many as 30 species of aphid.

The yellows viruses are often present in the leaf hopper vector for 10 days or more before the insect will transmit the virus. This may mean that the virus goes through an incubation period in the vector. The aster-yellows virus (19) and clover-club-leaf virus (21) have been shown to increase in the body of the insect vector. In the case of streak of corn (maize) it has been determined that certain strains of the vector are incapable of transmitting the virus. This has been shown to be due to the impermeability of the intestinal wall to the virus. If this wall is punctured at the proper time, the virus will pass into the blood stream, and the insect becomes capable of transferring it to the host plant.

Only rarely have leaf hoppers been shown to transmit mosaic viruses. Moreover, the yellows viruses are sometimes restricted to one or relatively few species of insect vectors.

The insect vector which transmits a virus to a given crop plant is not necessarily the species commonly destructive to that crop plant, and conversely the most common or most damaging species may not be vectors at all. The commonly destructive leaf hopper on potato which causes severe losses through hopperburn is *Empoasca fabae* Harris. It is not known to transmit any of the known numerous viruses of potato or any other virus. On the other hand the clover leaf hopper (*Aceratagallia sanguinolenta* Prov.), which visits potato but causes little or no damage to it, is a vector of the potato-yellow-dwarf virus.

Parasitic Phanerogams. Various species of *Cuscuta*, known collectively as dodder, live during a part of their life cycle as parasites upon other higher plants. By means of trailing stems they grow over the host plant and send haustoria into the leaves and stems of the latter. The haustoria make intimate connection with the cells of the vascular system. It was shown by Bennett (18) in 1940, and later by others, that certain viruses in the host plant may infect and become systemic in the dodder, without necessarily causing symptoms on the latter. As the dodder extends its stem to parasitize other plants, the virus may be transmitted to such plants through the newly formed penetrating haustoria. The dodder thus functions as the transmitting agent, although the virus cannot always be recovered from the dodder plant.

Up to the present, transmission of viruses by this means in nature has not been reported, or at least it is not regarded as common or important. Dodder has been used extensively in experimental work as a reservoir for a given

virus and as a transmitting agent. This procedure is particularly useful with viruses which are not transmitted mechanically, such as the beet-curly-top virus and the peach-yellows virus.

Soil Transmission. Relatively few of the plant viruses have been shown to pass from one crop season to another in the soil. This sometimes occurs in the case of the common-tobacco-mosaic virus, but it is not always clear whether its persistence is in soil or in undecayed infected plant refuse. Although the virus remains active for many years in extracted plant juice and in dried leaves, it does not usually persist in the soil more than a few months at the most. The virus of big vein of lettuce persists in the soil for more than a year, and the host is infected through the root system. The wheat-mosaic virus is known to remain infectious in the soil for as long a period as 6 years. It also invades the host through the root system, as does the tobacco-necrosis virus.

PROPERTIES OF VIRUSES

Physical Properties. Allard (1, 2, 5, 7, 8) was the first to use certain physical and chemical agents to study the nature of the tobacco-mosaic virus. He was concerned, at the moment, with determining whether or not the virus could be separated from the oxidizing enzymes in the plant extract. A few years later, Johnson, studying the same disease, became aware of the fact that more than one virus affected tobacco. While these diseases could be distinguished in part by their symptoms, it was obvious that not only did symptoms overlap but they also changed in appearance with variation in environment. Johnson (49) believed that a safer means of distinction was that based on differences between the viruses rather than on differences between the diseases they incited. He applied certain of the physical and chemical agents used by Allard such as reaction to heat, to dilution, to aging in vitro, and to formaldehyde, alcohol, and other chemicals. Viruses were found to differ markedly in the point at which they were inactivated by various agents. The tests have been used widely since, and in the modern characterization of mechanically transmitted viruses several of them are commonly applied. Those most extensively used are thermal inactivation (the constant temperature at which a virus extract is completely inactivated when exposed for 10 min.); aging in vitro (the number of hours or days at which a virus extract remains infectious when held at room temperature, usually 20°C.); dilution inactivation point (the degree of dilution of the virus extract with water or buffer solution at which infection no longer occurs). These are commonly referred to as physical properties. It is recognized that they are at best only crude qualitative indicators, but they are useful in placing unknown viruses in one or another virus group.

Host Range. The host range of a virus is used to aid in its characterization. Sometimes two viruses which incite similar symptoms on a given host can be distinguished by the fact that when they are inoculated to a group of plant species or varieties, some of the latter may be susceptible only to one virus, some only to the other. As an example, the typical-cucumber-mosaic virus and the typical-tobacco-mosaic virus incite symptoms on tobacco which are often indistinguishable. However, the cucumber virus affects cucumber systemically while the tobacco virus ordinarily does not. In earlier virus literature much importance was given to differences in host range in determining whether or not two viruses were distinct. Nowadays it is realized that different strains, which, on the basis of other property tests are known to belong to the same virus, may differ markedly in their host range. This means, of course, that this criterion is of minor importance in defining viruses, but it may be very useful in distinguishing between strains.

Serological Tests. Some years before the tobacco-mosaic virus was crystallized, evidence was presented by Purdy (69) (1929) that a partially purified virus extract exhibited antigenic properties whereby antiserum could be secured by injection of suitable animals. For instance, when juice extracted from a tobacco plant infected with the mosaic virus is injected into the blood of the rabbit in successive doses, the serum taken later from the animal causes flocculation when added in proper proportion to freshly extracted juice. Moreover when strains of the virus differing in various disease symptom patterns are tested against the same serum, they react similarly. This sensitive test, which is used to show similarities in proteins and bacteria, is likewise applicable to viruses. When a group of viruses give similar precipitin reactions to a given antiserum, they are regarded usually as strains of the same virus; when a virus gives no reaction, it is regarded as good evidence that it is distinct from the virus used to produce the antiserum. The serological test, then, is a useful tool in determining virus similarities and dissimilarities.

Cross-protection Tests. As early as 1906 Baur (13), working with infectious variegation in *Abutilon*, noted that if the leaves showing symptoms were continuously removed from the infected plant, the latter recovered in that it no longer showed symptoms. In 1924 Edgerton and Taggart (35) reported partial recovery of sugar cane affected with mosaic. They suggested that this might be a type of acquired immunity. In 1928 Wingard (96) reported that tobacco plants infected with the tobacco-ring-spot virus recovered from apparent symptoms and continued to remain healthy in appearance. However, the virus was recovered readily from such plants. Successive vegetative generations remained symptomless, but the virus was continuously present. This type of recovery has been found in various

other instances among plant viruses, and although it is sometimes referred to as "acquired immunity," it should not be interpreted as analogous with the common cases of acquired immunity in animals. The two major points of distinction are that immune antibodies have not been demonstrated and that the virus continues to multiply in the so-called immune host. Price (67) claims that in the case of tobacco ring spot, the amount of the virus in symptomless foliage was 10 to 20 per cent of that in leaves showing severe symptoms.

In 1931 Thung (88) showed that when tobacco showing symptoms of common tobacco mosaic was inoculated with Johnson's white-mosaic virus, the symptoms associated with the latter did not appear. If plants affected with white mosaic were inoculated with common-mosaic virus, the symptoms of the latter did not become evident. The same "protective" phenomenon was shown by Salaman (70) in 1933 for two strains of the potato-latent-mosaic virus. In 1934 Kunkel (54) noted a similar case with common-tobacco-mosaic virus and aucuba-mosaic virus on *Nicotiana sylvestris* Speggazini & Comes. The latter virus usually causes only local lesions on the inoculated leaves of this host. When the host was first inoculated with the common-mosaic virus and when leaves showing systemic symptoms were inoculated with the aucuba-mosaic strain of the virus, the usual local lesions did not appear. Numerous other cases similar to those cited above have now been recorded. It is generally assumed that when one virus, preceding another in the tissue, prevents or markedly impairs increase of a virus subsequently introduced, it is an indication that the two viruses are closely related and are probably strains of a single virus. The phenomenon is most striking when one virus is allowed to become well established systemically before a related strain is introduced. If the two related strains are introduced simultaneously, it is more likely that both will multiply, and mixed symptoms will appear if both are systemic. There is ample evidence that all related strains do not protect completely against each other. Matthews (61) studied 10 strains of potato-latent-mosaic virus, two of which only partially protected against the others. Two each had an antigenic fraction specific to the respective strain, and they shared a fraction not shared by the other eight. The cross-protection test is only an indicative one which may be used in conjunction with others in the characterization of a virus.

Purified Virus Proteins. Since the preparation of crystalline tobacco-mosaic-virus protein was announced by Stanley (78) in 1935, nearly a dozen plant viruses have been prepared in more or less purified condition. The ultracentrifuge, the electrophoretic apparatus, X-ray diffraction, and the electron microscope have aided greatly in intensive physicochemical studies of the nature of virus particles. It is not feasible to review the rapid progress in this field here. The student is referred to such compilations as those

by Bawden (14) and Cook (26) for details. All viruses crystallized so far are nucleoproteins. The virus particles of tobacco mosaic are about 15 mμ in diameter, and they vary in length (Fig. 156). While there are differences in opinions as to the range of length of the infective particle, it is the contention of some that when the length is about 280 mμ, the particles may aggregate by becoming attached end to end, sometimes to form longer strands. The aggregates may have a relation to the crystalloid inclusion bodies found in infected host cells. If the particles are broken into lengths below 280 mμ by supersonic waves, they retain their serological activity but lose most of their disease-inciting property.

The molecular weight of the tobacco-mosaic molecule is about 40,000,000, which is about 2,000,000 times that of water. The molecular weight of the tomato-bushy-stunt molecule is between 10,000,000 and 11,000,000.

VIRUS STRAINS AND VIRUS MUTATION

Beginning about 1925 investigators of plant viruses began to recognize strain differences. McKinney (56) was the first to report the separation of a yellow-mosaic strain of common tobacco mosaic from the ordinary type. This was done by picking small bits of tissue from bright yellow areas in infected leaves. Since then strains from many viruses have been isolated by various modifications of this method. Koch (51) separated ring-spot and mottle strains of potato-latent-mosaic virus by taking advantage of the fact that one moved more rapidly than the other through the host from the point of inoculation to the growing tip. This technique has also been used frequently by others. Jensen (47) isolated 26 yellow strains of tobacco-mosaic virus. Price (67) isolated one or more yellow strains of cucumber mosaic. In most viruses studied intensively one or more strains have now been recognized. Sometimes strains differ in virulence and in symptoms which they incite and are very similar in host range and in physical properties. In some cases, as in numerous strains of cucumber-mosaic virus, the chief differences are in host range.

The origin of virus strains is of interest. McKinney (59) suggested that strains arise by mutation, and Price (67) in his study of cucumber mosaic came to the same conclusion. While this is difficult to prove, there is much evidence pointing to mutation as a common phenomenon in plant viruses. As yet mutation has not been shown to occur in vitro. The nature of virus mutation within the host cell is still as much a riddle as is the nature of multiplication of the virus itself. Various physical studies of related strains, particularly in tobacco mosaic, have yielded no measurable differences in physical properties and in the nature of the particles exhibited by use of the electron microscope. The work of Knight (50) on the microbiological assay of amino-acid components of tobacco-virus strains has

shown that they do vary in the number and relative percentage of the acids present. It is quite possible that certain of the radicals of the virus protein which have to do with virulence and other properties associated with host pathology may change (mutate) while those radicals which have to do with antigen properties do not. Thus a wide range in virulence and in host selectivity may exist between strains, and, at the same time, a high degree of uniformity in serological reaction may prevail.

SYNERGISTIC EFFECTS OF VIRUSES

In 1925 Dickson (27) showed that the severe disease of greenhouse tomato plants known as winter streak was due to the combined effect of the tobacco-mosaic virus and a potato-mosaic virus. Vanterpool (90) confirmed these results. It was shown later that American potato varieties usually contain the latent-mosaic virus and that this is the important component in the combination with the tobacco-mosaic virus. These two viruses when present separately in tomato incite relatively mild effects, but when both are present, the effect is very severe. The combined action of two infective agencies is referred to as a **synergistic** effect. Valleau and Johnson (89) pointed out that the streak symptoms might be provoked by the synergistic effect of several virus combinations. There are numerous cases now known in which virus diseases may be the result of synergism of two or more viruses. We have, in such cases, the counterpart of cross-protection, in that unrelated viruses do not interfere with one another but, on the contrary, often enhance each other's effect upon the host.

DEVELOPMENT OF CONCEPTS OF THE NATURE OF VIRUSES

There are records to show that what we now know to be virus diseases were described many years before their causal factors were studied. Breaking of tulips was mentioned as early as 1576. Transmission of leaf variegations from scion to stock of woody plants was recorded as early as 1700. Degeneration of potatoes was known in some parts of Europe by 1770 and was severe in some areas by 1775; Anderson, writing in 1778, showed it to be transmitted with the seed tuber and likened it to smallpox in the nature of its infectiveness. Peach yellows was recognized as early as 1791 in the eastern United States. Tobacco mosaic was recognized by Swieten in Holland in 1857. Adolph Mayer (62), a Dutch investigator working with this disease in the last-named country, was the first to point out, in 1886, that it was readily transmissible and infectious. He endeavored to find a causative organism in the form of a fungus or bacterium but failed in this attempt. He believed that he had ruled out faulty nutrition, nematodes, and environment as causal factors. Nevertheless, he demonstrated, for the first time, that the sap of diseased plants contained something which, when the juice

was applied to wounds on leaves of healthy plants, caused the young growing parts to develop the characteristic symptoms of the disease. Mayer thought that probably a bacterium was the incitant but that he failed to isolate it by the technique he used. It was, indeed, only 2 years after the announcement of the poured-plate method of isolation of bacteria by Koch.

At about this time Erwin F. Smith, a member of the U.S. Department of Agriculture, began a study of peach yellows, which was recognized as a disease of declining peach trees in the eastern United States and in Michigan. He published his first note on the trouble in 1888. A full report was given in 1891 (71) on this and another virus disease which was called peach rosette. Smith believed that these virus diseases had some points in common with tobacco mosaic of Mayer. One was the fact that no microorganisms were isolated and identified as causal agents. Another was their transmissibility in spite of the lack of an apparent causal factor. Yellows and rosette differed from tobacco mosaic, however, in that they could not be transmitted as readily. The extraction of juice from diseased plants and inoculation into healthy trees did not result in infection of the latter. The only way in which transmission was secured was by grafting buds from infected trees into healthy trees. A year later Iwanowski (45) demonstrated that the tobacco-mosaic virus would pass a bacteria-proof filter, and thereby he distinguished, for the first time, this type of infectious agency from bacteria and from fungi. This was confirmed in 1898 by Beijerinck (17), who showed further that the tobacco virus will diffuse through an agar membrane. Koning (52) in Holland added further confirmation in 1899. In 1898 Loeffler and Frosch were the first to show a virus of animal origin to be filtrable when they demonstrated that the foot-and-mouth disease of cattle was incited by an entity which passed a bacteria-proof filter.

By the end of the century, then, it was established that a certain type of plant disease, characterized in a general way by chlorotic or mosaic symptoms on the foliage, was infectious but that the causal agent was not isolated according to the methods which had rapidly evolved during the two previous decades for the cultivation of pathogens on artificial media. To be sure, numerous recognized pathogenic fungi, notably the rusts, had not yielded to artificial cultivation either. It thus remained for another 35 years a question whether the virus diseases, which became recognized in large numbers during that period, were incited by living or nonliving entities.

In 1902, Woods (97), working in the U. S. Department of Agriculture, proposed a theory that enzymes were responsible for the diseased condition in tobacco plants affected with mosaic. He found an excess of oxidizing enzymes in the diseased tissue, which he claimed to be characteristic of tissue of variegated plants. He believed that the disease "is the result

of defective nutrition of the young and rapidly growing cells, due to lack of elaborated nitrogenous reserve food accompanied by an abnormal increase in activity of the oxidizing enzymes in the diseased cells. The unusual activity of the enzyme prevents the proper elaboration of reserve food, so that a plant once diseased seldom recovers." Heintzel (37) in Germany came to the conclusion, independent of Woods, that enzymes were responsible for the disease. Hunger (44), also in Germany, did not accept the enzyme theory but considered that unfavorable conditions of growth produced specific toxins within the plant which led to the appearance of the disease.

Baur (12) in Germany (1904) showed that the variegations in some clones of woody plants were noninfectious while others, such as the chlorosis of *Abutilon*, were transmitted to nonaffected plants by grafting. He believed that the pathogenic agent in the latter case was nonliving but comparable to the incitant of peach-yellows disease as described by Smith.

The importance of tobacco mosaic in the crop of the Connecticut River valley had continued to attract the attention of investigators since Sturgis (85) reported upon the occurrence of the disease in 1899. Clinton (25) showed that the virus would infect tomato and incite a similar disease and that the virus from tomato would reinfect tobacco.

In 1914 Allard (1–10) of the U. S. Department of Agriculture published the first of a series of papers which added many new facts regarding the disease and challenged the enzyme theory. He extended the host range to several species of *Nicotiana*, *Petunia*, *Physalis*, *Datura*, and *Solanum* and to *Hyoscyamus niger* L. and *Capsicum frutescens* L. The virus was recovered from all parts of the tobacco plant except the mature seed. A series of experiments was carried out in which the effect of various physical and chemical factors upon the infectivity of the sap extracted from diseased tobacco plants was determined. No reduction in infectivity was noted at a dilution of 1–1,000 in water, but at 1–10,000 reduction was evident, and at 1–1,000,000 only an occasional plant was infected. He noticed some reduction in infectivity when juice was passed through a Berkefeld filter, and when he used a Livingston atmometer, the filtrate was no longer infective but gave a strong peroxidase reaction. He removed the virus by filtering through talc, but the filtrate again gave a strong test for the enzyme. He destroyed the enzyme in the infective juice with hydrogen peroxide, but the virus remained active. The virus was destroyed by 1 part of formaldehyde to 1,000 of juice, but peroxidase was still intense. The juice was rendered noninfective at 90°C. When heated for 10 min. at 85° on 4 successive days, no peroxidase reaction was had, but the juice was still infective. Allard believed he had completely disproved that oxidizing enzymes had any causal relation to the disease. He believed

that the virus must be regarded as a parasite and expressed himself as follows:

> In the writer's experience all evidence at hand indicates that the mosaic disease of tobacco is dependent upon a specific pathogenic agent which must be introduced into healthy plants from without before the disease can arise. That this pathogenic entity is highly infectious and is in some manner reproduced within the plant are established facts. If these facts are interpreted according to those fundamental principles upon which all our scientific conceptions in pathology and biology are based, that infectious diseases are associated with parasitism and that self-reproduction is a characteristic of living things alone, it must be admitted that the pathogenic agents responsible for the mosaic disease of tobacco must be parasites. If from the facts stated above it is held that non-living chemical substances such as enzymes or toxins engender the disease, our fundamental biological conceptions no longer hold true.

It is of interest to note here that although Allard maintained that the virus was a living entity, he later found that it was very tolerant to numerous chemicals, *e.g.*, mercuric chloride and phenol, which are strongly germicidal.

About this time attention was directed by a few to the possibility of insect relations to this type of disease. In 1900 Walter Reed transmitted yellow fever in man by means of the mosquito, and it had recently been shown, along with certain other diseases of man and animals, to be incited by a filtrable virus. In 1895 Takata in Japan transmitted the infectious entity of rice stunt by means of the leaf hopper *Deltocephalus dorsalis* Motsch. In 1908 Takami showed that the leaf hoppers were noninfective until they had fed upon diseased rice plants. In 1906 Ball (11), working with curly top of sugar beet in Utah, associated the latter disease with the occurrence of a species of leaf hopper. Ball, however, attributed curly top to the direct injury caused by the hopper and did not consider that a virus entity was concerned. The exacting proof of this was provided by Boncquet and Hartung in 1915 (22). The first thoroughgoing study of insect transmission of a plant virus was reported in 1914 by Allard (1) of the U.S. Department of Agriculture. He used the tobacco-mosaic virus, although there is reason to believe now that he had a mixture of tobacco and cucumber-mosiac viruses and that the observations on insect transmission apply to the latter. Allard demonstrated that the tobacco aphid (*Macrosiphum tabaci* Perg.) and the peach aphid (*Myzus persicae* Sulz.), when fed for a day or more on infected plants and transferred to feed on a healthy plant, promptly inoculated the latter, in which the normal course of disease development followed.

Iwanowski (46), continuing his study of tobacco mosaic, had described in 1903 certain unusual intracellular bodies in the tissues of diseased plants.

One type was amoeboid; the other was in the form of crystalline plates. This work attracted little attention at the time although cell inclusions had been described earlier with *contagiosum molluscum* by Henderson (1841), with fowl pox by Rivolta (1869), and with vaccinia by Guarnieri (1894). The Negri bodies described in connection with rabies, a virus disease of man and animals, received much attention when described almost simultaneously with the inclusion bodies of tobacco mosaic by Iwanowski. The latter did not believe the bodies were causal organisms since they could not be expected to pass through filters. Kunkel (53) described similar intracellular bodies in connection with corn mosaic in 1921 and implied that they might have a causal relation. It was thought by some that a naked protoplasmic organism, such as certain of these bodies seemed to be, might pass porous filters and still exhibit all the other properties ascribed to infectious tobacco juice, particularly if it could be shown to have a heat-resistant stage with an extended longevity. Nelson (65) in 1922 described bodies of a different nature in the phloem of mosaic-infected bean plants and propounded a protozoan theory of causation. This idea was promptly refuted when numerous workers were able to show that Nelson's bodies were the normal, elongate, more or less protozoid shaped nuclei characteristic of the sieve tubes of many species of higher plants. The theories based on microscopically visible intracellular entities as causal factors rather promptly fell by the wayside, although such bodies have been shown repeatedly to be associated with infected tissues of some viruses (16, 38, 40).

In the meantime some investigators continued to direct their researches toward the physicochemical nature of the virus. Duggar and Karrer (30) in 1921 published an important paper in which they presented new information as to the size of the infective particles. They used a series of porcelain and collodion filters which could be calibrated as to the size of the openings. They found the infective particles to pass some of the filters and not others. They estimated that the infective particle was in the size range of hemoglobin and was approximately 30 mμ in diameter. This was the first exacting attempt to delimit the size of the particle. It showed that it was much smaller than the smallest known bacterium and in the size range of the larger molecules. Duggar (28) in 1926 became convinced that the "infectious agency is a particle of almost inconceivably small size, certainly too small to represent an organism with the usual characteristics." He expressed his viewpoint further as follows:

> The difficulty in conceiving of this disease agency as anything but a living organism of the usual type is due chiefly, I think, to the indefiniteness with which we are faced. We know that the virus has the power of multiplication within the host, and this capacity for reproduction, in general, is associated with a living organism, and along with this capacity there are other properties which seem to satisfy the

requirements of many criteria of life, such criteria as might seem properly to apply to theoretically simple life.

Summarizing, it appears that with the facts already reviewed we must either consider that this virus is not a living thing at all or that it constitutes a kind of living thing of which we have had previously no very definite conception, unless the extensive work on the bacteriophage may be considered analagous.

To satisfy the experiments presented, it would seem almost necessary to assume a low order of correlation, since this would be the only possibility in a body of colloidal dimensions. The other alternative is the assumption of a chemical substance capable of stimulating the protoplasm of the infected host to reproduce the same material, an assumption that has been made twice or three times earlier; but this is an assumption which has as a basis, it would seem to me, far more uncertain analogies.

I can only look upon this problem of the nature of the mosaic virus as one which may stimulate to renewed efforts investigation in an almost unworked field, that of the lower orders of life, which it would seem must exist in many other habitats or conditions somewhere.

Sooner or later we must more frequently ask what, if any, are the various connecting links between complex organic compounds and the simplest known living organisms. Surely it is not possible to conceive of bacteria or protozoa as the kinds of living things that would emerge directly from a colloidal nitrogenous matrix. The elaborate correlations existing in the cell as we now know it are stupendous, and it would seem that there must be some simpler life somewhere. The soil and water areas alike should offer the opportunities needed.

Possibly we can know nothing of these because of the difficult technique involved but surely we should not neglect to explore any paths that may lead to fields fruitful in newer facts concerning life. With material at hand so interesting as virus diseases of plants, pathologists and physiologists have an opportunity and a responsibility such as is seldom afforded. Pasteur, as we all know, is commonly supposed to have disproved the idea of the spontaneous generation of life. As students of living beings and of their development, however, we know that he disproved no such thing, but rather he proved the known forms of life did not originate in a brief space of time in a sterilized nutrient fluid. This fact is of incalculable value and has prevented many false theories. Nevertheless, the proof which he offered would not operate to deter investigations that may take us nearer to an understanding of lower orders of life whether or not that may be synonymous with a higher order of chemical substances.

Mulvania (63, 64) (1926) showed that rabbit blood inactivated the virus. He dialyzed the virus through some of the collodion membranes which were made up in a series of grades of porosity. He was of the opinion that the virus was of "non-vitalistic origin" and that it "may be conceived as a product of the plant's own metabolic activity under the influence of some disturbing factor introduced into the plant body, namely the virus produced by other plants similarly affected. Thus we invoke the principle of auto-catalysis."

It became apparent to Duggar that, regardless of the vital or nonvital nature of the virus, it showed properties in common with colloidal particles, or macromolecules. He pointed out in 1926 (28) that when one attempted to free crude infectious tobacco juice from extraneous material and retain the virus in the clarified liquid, the virus was apparently adsorbed by some of the clarifying agents. He had noted that when pokeweed juice was added to infectious tobacco juice at the ratio of 2 volumes to 1, the infectivity was destroyed. In lesser amounts the degree of reduction in infectivity was in proportion to the ratio of pokeweed juice added; at the ratio of 1-5 it had no effect. This appeared to be an adsorption phenomenon also. Calcium chloride gave a precipitate which carried down the virus but released it upon inoculation. Charcoal rendered the juice noninfective, but different charcoals of animal and plant origin acted differently in this regard. In 1927 Brewer *et al.* (23) clarified the infectious juice by passing it through a sintered-glass filter, washing the residue on the filter until the filtrate was colorless. When the residue was suspended in water, it was found to be infectious. By a second and more effective method they centrifuged the crude juice at 35,000 r.p.m. and passed the fairly clear supernatant liquid through a powdered-charcoal filter. The charcoal adsorbed the virus, and after thorough washing it was suspended in water, when enough of the virus was eluted to render the supernatant clear liquid infectious. In later experiments (24) (1930), they centrifuged the crude juice, discarded the liquid, resuspended the residue, centrifuged again, and then cleared and decolorized the liquid with alumina gel, acid in reaction. This infective clarified liquid remained active up to pH 2.46, but at 7.5 to 8.5 a precipitate formed which rendered the liquid noninfective. However, when the reaction was readjusted to below 7.5 the virus again became infective. McKinney (57, 58) worked simultaneously upon the use of centrifugation to purify virus extracts.

Vinson and Petre (91-95) (1929–1931) precipitated the virus from crude infective juice with an aqueous solution of safrinine, which held the virus in an inactive state until it was removed by amyl alcohol. The virus was also salted out with ammonium sulfate and magnesium sulfate. They found that the addition of 2 volumes of acetone or alcohol to 1 volume of juice threw down a precipitate which contained practically all of the virus. Dilute lead acetate or barium acetate threw down phosphate, sulfate, and most of the protein and pigment in the juice without removing or changing the virus. They concentrated the cleared juice *in vacuo* and brought the temperature to about 0° C., when 2 volumes of acetone at 15° were added. A precipitate formed which contained only about 10 per cent of the solids of the original juice and apparently all of the virus. By further refinement of methods they first cleared the juice with dilute basic lead acetate and

then precipitated the virus with a stronger solution of neutral lead acetate. The virus was released from the precipitate by suspension in a normal solution of primary potassium orthophosphate. *The suspension then contained only about 1 per cent of the original total solids and was still highly infectious.* The infectivity of the juice was increased by the presence of inactive substances such as talc and norite. *They found the percentage of nitrogen to increase as the total solids decreased, and in the more highly purified fractions the nitrogen content was close to that of the simple proteins.* They secured crystals from some of the preparations which were moderately active but did not think that the crystals represented the pure virus. They considered that the behavior of the virus in the purified preparations paralleled closely that of some of the enzymes. They stated that "of the various theories, therefore, put forward as to the nature of the virus, the enzymic one advanced by Woods and further emphasized by Freiburg (36) fits in with the experimental results here reported." Stanley (1935) (77) later showed that the yield of virus in the lead acetate precipitate was influenced greatly by the pH of the medium. The optimum pH for the lead subacetate precipitation was about pH 9, for neutral lead subacetate precipitation about pH 5.5, and for elution of the virus from the precipitate about pH 7.

The investigations up to this time were hampered by the lack of a means of accurately estimating the concentration of the virus in various preparations. Determination of concentration of virus was based upon the percentage of plants infected. Such data, to be significant, would have to represent an enormous number of plants. A very important boon to this line of investigation was the local-lesion method developed by Holmes (42, 43) and announced in 1929. This consisted in using leaves of hosts which responded to inoculation by developing local necrotic spots. Holmes showed that the number of spots bore a relation to the concentration of the virus in the inoculum and that the method could be used as a measure of concentration. All later investigators have relied heavily upon this method. *Nicotiana glutinosa*, the F_1 hybrid of *N. tabacum* crossed with *N. glutinosa*, and some varieties of bean (*Phaseolus vulgaris* L.) are suitable hosts for the assay of liquids containing the tobacco-mosaic virus.

Other methods of purification of virus preparations followed. Duggar (29) found, as had Vinson and Petre, that freezing the tissue before extraction resulted in a clearer juice. Various adsorbents were used to clarify the juice. Norite and various grades of Nuchar gave rapid decolorization of tobacco-juice extract but also adsorbed the virus to some extent. Celite was found to be a satisfactory adsorbent which, after being mixed with crude juice and allowed to stand, was removed by centifuging, leaving a clear infectious liquid. Burt Johnson (1934) (48) studied methods of increasing the concentration of the virus in the clarified liquid. Am-

monium sulfate, when added to the infectious liquid, precipitated the virus most completely with least inactivating effect. The salt was then removed by dialysis, leaving the virus in suspension. The suspension was concentrated by evaporation at room temperature to a sirupy consistency, resuspended in water, and centrifuged. The supernatant, slightly clouded liquid showed a distinctly higher concentration of the virus than did the original infectious juice.

In 1933 Takahashi and Rawlins (86, 87) published a very significant series of observations. They found that the juice extracted from frozen tissue of infected tobacco, when exposed to polarized light, exhibited the phenomenon known as stream double refraction. This had been shown to be characteristic of sols containing rod-shaped colloid particles. They found that the juice of healthy plants prepared in the same way showed no stream double refraction. When juice of healthy plants extracted without previous freezing of the tissue was used, there was some stream double refraction but much less than in juice of diseased plants so prepared. When juices of healthy and of diseased plants were subjected to the safranine - Lloyd's reagent treatment of Vinson, the purified preparation of healthy juice showed no stream double refraction while the preparation of infected juice could usually be diluted 200 times before double refraction disappeared. These results indicated that the virus particles were rod-shaped, a fact which was fully demonstrated by later work of others.

Duggar and Hollaender (34, 39) irradiated the tobacco-mosaic virus and certain of the bacteria with monochromatic light of different wave lengths. They first compared the virus with *Serratia marcescens* Bizio, using 12 spectral lines or groups of lines in the range λ 2537 to 6120 Å. The inactivation of the virus was confined to wave lengths shorter than about λ 3100 Å. The energy required at λ 3100 Å. was more than 100 times that required at λ 2652 Å. At the latter wave length the virus was about 200 times as resistant as the bacteria. The virus was also found to be much more resistant than the spore stages of *Bacillus subtilis* Cohn and *B. megatherium* DBy. When a more highly purified preparation of the virus was used in comparison with *Escherichia coli* (Migula) Castellani & Chambers, the virus was found to be 40 times as resistant as the bacteria at λ 2650 Å., but only 3.1 times as much energy was required to inactivate 50 per cent of the virus as to kill 50 per cent of the bacteria. The inactivation curve of the virus was suggestive of those for urease and pepsin.

Vinson and Petre (94) and Lojkin and Vinson (55) studied the effects of enzyme preparations on the infectivity of the tobacco-mosaic virus. They suggested that the action of trypsin was probably due to its hydrolytic effect upon the virus. Matsumoto and Somazawa (60) found that the antigenic properties of the virus were partly destroyed by trypsin. Vinson

(93) later found that although trypsin inactivated the virus, the infectivity was restored in part when the mixture was heated. Working with a highly purified preparation of trypsin supplied by Northrup (66), Stanley (72) reported that the decrease in infectivity caused by trypsin could not be due to its proteolytic activity because the action was immediate; it took place at a pH at which trypsin was proteolytically inactive; and the infectivity was restored by heat and dilution and by digestion or removal of trypsin. His results indicated that there was no combination of virus with trypsin. He believed that the reduction in infectivity was due to the effect of the enzyme on the plant at the time of inoculation. The last concept has not been upheld by the work of others. Stanley pointed out that additions of the proteins globin and trypsinogen, which possess no proteolytic activity, had the same effect as trypsin. When pepsin was used (73), there was no inactivation of the virus until the pH was adjusted to pH 4 or less. At pH 3 and 37°C. inactivation took place in proportion to the concentration of the pepsin and the time of digestion. Reactivation of the virus was not attained. The evidence justified the conclusion that the inactivation was due to the proteolytic activity of the enzyme and suggested that the "virus of tobacco mosaic is a protein, or very closely associated with a protein, which may be hydrolyzed with pepsin." The effect of hydrogen-ion concentration on the virus showed that it was irreversibly inactivated only at pH 2 or less and pH 11 or above (75). The effect of 110 chemicals was investigated (76). With the exception of brucine, the only chemicals which affected the virus directly were oxidizing agents, protein-precipitating agents, and materials causing a hydrogen-ion concentration known to inactivate the virus. Stanley stated:

> The fact that this virus is precipitated by so many protein-precipitating agents is an indication that it is protein in nature. Proteins are relatively inert chemically at or near their isoelectric point, and are more reactive at hydrogen-ion concentration removed from the isoelectric point. Mercuric chloride was found to affect virus to a markedly less extent at pH 3, 4, and 5 than at pH 6, 7, and 8. This suggests that this virus may be a protein having an isoelectric point near pH 4.5, thus rendering it inactive chemically toward mercuric chloride near pH 4.5, but allowing reaction with mercuric chloride at pH 6, 7, or 8. The fact that tobacco-mosaic virus was found to be unaffected over long periods of time by concentrations of mercuric chloride known to be germicidal, indicates that it is not a bacterial organism. As a whole, the results are in harmony with the conception that this virus is a protein.

By 1935 it was evident that most researches on the nature of the tobacco-mosaic virus were tending strongly toward investigation of it by chemical methods. Both physical and chemical tests yielded more and more evidence that the virus was a particle distinct from any known living entity and comparable in many ways to larger molecules or colloidal particles.

The techniques were being rapidly improved whereby the virus extract could be cleared of inert extraneous material. It could be precipitated and eluted. It was adsorbed and temporarily inactivated by a variety of substances. The purest preparations were relatively high in nitrogen. Pepsin destroyed the virus activity. By this time Sumner and Northrup had prepared some of the enzymes in very pure form and showed that they were proteins. All signs pointed to the virus as a protein. Nevertheless the biological world was startled when Stanley (78) announced in *Science* of June 28, 1935, that he had isolated a crystalline protein possessing the properties of the tobacco-mosaic virus. He stated: "Tobacco-mosaic virus is regarded as an autocatalytic protein which, for the present, may be assumed to require the presence of living cells for multiplication."

In the succeeding 5 years protein crystals were isolated from preparations of several plant viruses. These preparations were infectious and capable of producing the disease concerned upon inoculation into the respective host. Stanley at first erred in his analysis and identification of the tobacco-virus protein, calling it a globulin protein. The work of Bawden and Pirie in England served to show that this and other identified virus proteins are nucleoproteins. At the present time the virus entities are regarded by many investigators as nonliving filtrable proteins which are to be found in preponderance over normal proteins in virus-diseased plants. One theory as to their production supposes that autocatalytic action goes on when a virus particle is introduced into the normal healthy plant with the result that a considerable percentage of the protein formed is virus protein rather than normal protein. The relation of the intracellular bodies to this concept is not entirely clear, but certain of them may possibly be aggregates of protein crystals *in situ* in the cell.

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TOBACCO AND TOMATO MOSAIC

The early history of the tobacco mosaic has already been discussed (p. 35). It attracted first attention in the United States in the Connecticut River valley and has since been recorded in many tobacco-growing areas throughout the world. Clinton in Connecticut was the first to show that the virus infects tomato, and the disease is now recognized as one of the most common on that crop. Allard transmitted the virus to several species of the Solanaceae including pepper, petunia, henbane, jimson weed, black nightshade, ground cherry, and several species of *Nicotiana*. Grant (7) explored the host range of the virus and found 29 susceptible species in 14 widely separated families outside of the Solanaceae. The list includes beet, spinach, phlox, foxglove, zinnia, buckwheat, snapdragon, white mustard, turnip, Kenilworth ivy, and tassel flower. Holmes (10) extended the host range still further.

Symptoms. In young tobacco plants the first visible symptom is a downward curling and distortion of the youngest leaves accompanied by chlorosis. As the leaves enlarge, abnormally dark green spots appear which develop into irregular crumpled blister-like areas while the remainder of the tissue becomes more and more chlorotic (Fig. 155). Various degrees of leaf malformation follow, and some leaves show only a mild diffuse mottle. As new leaves unfold, they continue to show the typical range of symptoms. In pink-flowered forms the normal color pattern of the flower is disturbed by interspersion of light-colored areas or complete suppression of color. Old leaves may show no mottle but may contain large yellowish

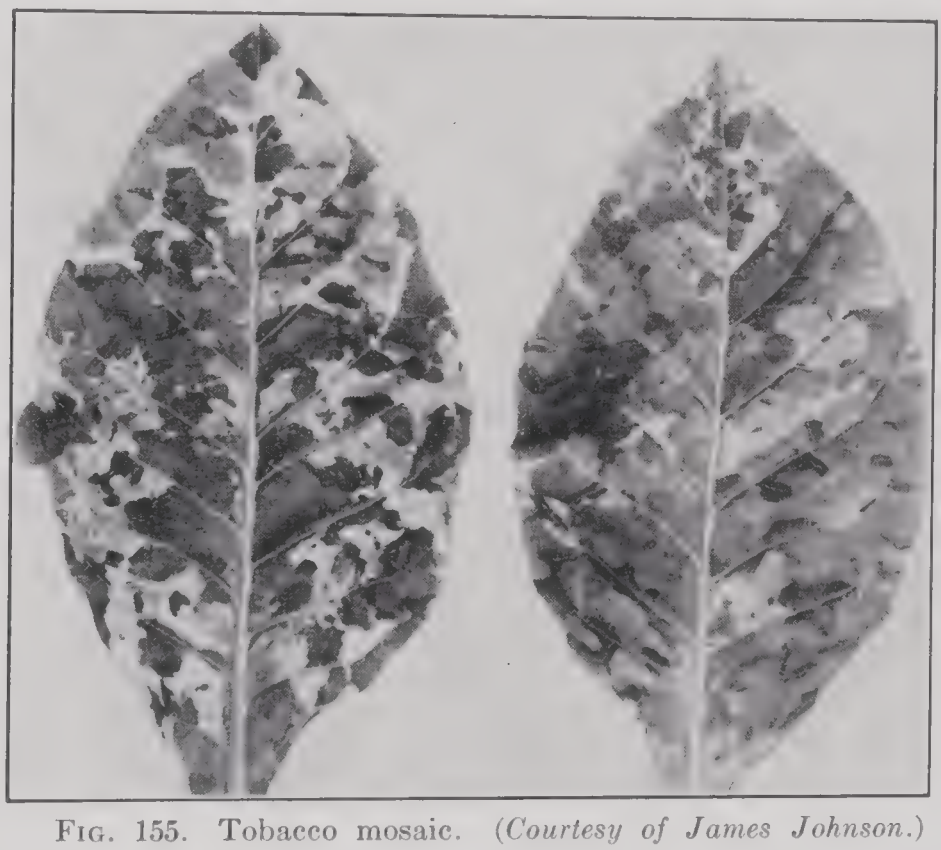

FIG. 155. Tobacco mosaic. (*Courtesy of James Johnson.*)

blotches or scattered necrotic spots, symptoms often referred to by growers as "rust." Plants are stunted in various degrees. With some strains of the virus extensive necrosis occurs, and with others the chlorotic areas are more intensely yellow and more extensive in size. The usual symptoms on tobacco are very similar to those on the same host incited by typical-cucumber-mosaic virus, and the two diseases on tobacco are often confused.

The typical appearance in tomato is a mild mottle of the foliage. With some strains of the virus yellow chlorosis is predominant. In other cases severe strains cause necrosis of leaf and stem (streak) and necrosis and distortion of the fruits. This phase is commonly called single-virus streak. Quite similar streak symptoms are incited by the synergistic effect of latent-

mosaic virus of potato and any strain of the tobacco-mosaic virus or by the former in association with typical cucumber mosaic (24, 25).

The Virus.

Typical-tobacco-mosaic virus.
Tobacco virus 1.
Nicotiana virus 1 K. M. Sm.
Marmor tabaci Holmes.

FIG. 156. Particles of purified tobacco-mosaic virus as shown by electron microscopy. (*Courtesy of M. A. Stahmann.*)

The virus is distinctive in that it remains active in extracted host plant juice up to 25 years, longer than any other known virus. It is infectious in dilutions up to 1–1,000,000 and withstands heating up to about 90°C. for 10 min. The virus protein was first described by Stanley (22) in 1935 (Fig. 156). An immense amount of fundamental work on the physico-chemical properties of the virus has been carried out since. For a summary of these studies refer to Bawden (1) and to Stanley (22).

McKinney (15) in 1926 first isolated a yellow-mosaic strain from tissue taken from yellow spots in normally affected leaves. He (16) later suggested that such strains arose by mutation, and further work by others tends to confirm this view. Jensen later (12) described 12 strains from a total of 55 isolates which varied in symptoms incited.

Many investigators since Iwanowski have studied the cell inclusions characteristic of tissue infected with the tobacco-mosaic virus. Two types of intracellular inclusions are found. The first of these, often referred to as X bodies, are amorphous, protoplasmic, more or less vacuolate bodies (Fig. 157). The other is striate material consisting of crystalline plates

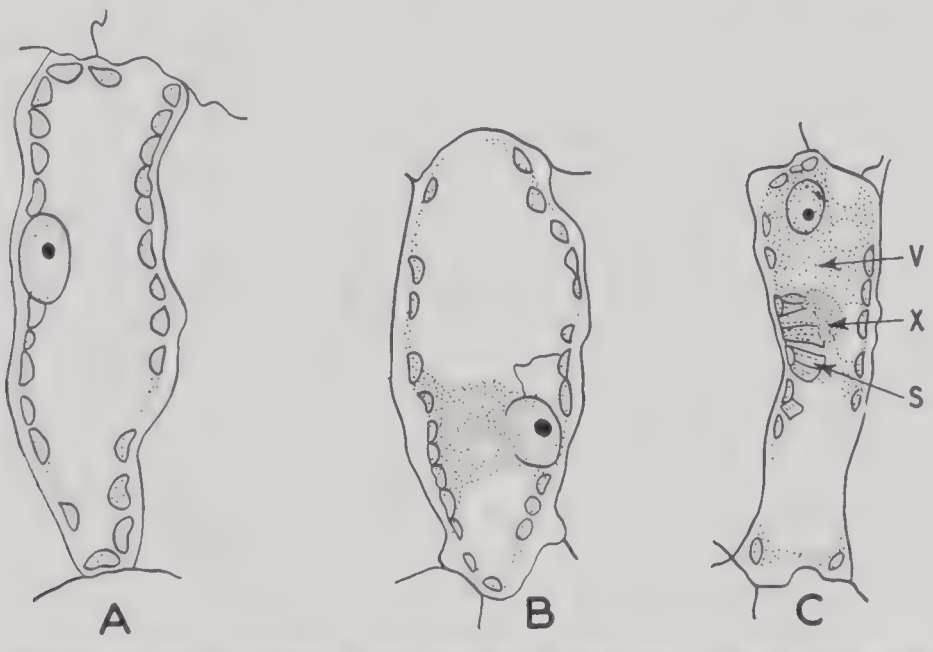

FIG. 157. Intracellular bodies. *A*, palisade cell of potato; *B*, palisade cell of potato infected with the mild-mosaic virus, showing vacuolate material next to the nucleus; *C*, palisade cell of potato infected by the common-tobacco-mosaic virus, showing inclusion body (X body) (*x*), striate material (*s*), and vacuolate material (*v*). (*After Hoggan.*)

which give positive protein reactions (2, 8). Attempts to remove these bodies by micromanipulative methods have not been successful (21). The striate material has some resemblance to the purified virus-protein crystals, and it sometimes appears as the X bodies disappear. It is not likely that crystalline inclusions are deposits of the pure virus, more probably they are combinations of the latter with some other constituents of the host cell (2).

Disease Cycle. Sources of primary inoculum consist of debris of infected plants which remains in the soil, tobacco refuse from warehouses, and certain infected manufactured tobacco products contaminating hands of workers (13, 14). Johnson (13) in a series of tests found 67 per cent of cigars, 81 per cent of cigarettes, and 62 per cent of pipe tobaccos to carry

the virus. Manufactured chewing tobacco and snuff carried little or no virus, probably on account of the high temperatures to which the tobacco is exposed in manufacturing.

Dissemination from plant to plant takes place through mechanical transmission. One means is through handling of tobacco and tomato plants at transplanting. Other operations which afford ready means of transmission are suckering of tobacco plants and suckering and training of tomatoes to a single stem in hothouse production of the latter crop. Contact by man, animals and machinery during cultivation of the crops is another means of spread of the virus. The virus is not known to be transmitted by insects from tobacco to tobacco. However, the false potato aphid, *Myzus pseudosolani* Theob., transmits the virus readily from tomato to tomato and tobacco. The aphid species, *M. circumflexus* Burkt. and *Macrosiphum solanifolii* Ashm., transmits it infrequently while the peach aphid, *Myzus persicae* Sulz., is incapable of transmitting this virus (9).

Varietal Resistance. Two types of resistance in tobacco are known. One type is that found in the Ambalema variety discovered by Nolla (20) in Colombia, in which the virus increases but at a slow rate and symptoms are very mild. The other type is the local necrotic reaction found in *Nicotiana glutinosa* L. This character is dominant in the F_1 hybrid of *N. glutinosa* × *N. tabacum* L., but since the plants are self-sterile, special means must be employed to transfer the necrotic character to *N. tabacum* (3, 4, 6, 11, 17–19, 23).

Control. The first precaution in control is to avoid infested soil and to avoid the use of tobacco refuse on land on which tobacco or tomato is to be grown. Seedbeds should be steam sterilized (see p. 661) and well removed from tobacco warehouses and from greenhouses in which tomatoes are growing. Care should always be exercised to avoid contamination of hands with the virus from tobacco products and above all to avoid handling potatoes or cucumbers before staking or suckering tomatoes or tobacco. Thorough washing with soapy water is an effective practical means of cleansing hands between operations.

Some progress has been made in the direction of breeding of resistant varieties of tobacco and tomato (5), and some commercial mosaic-resistant varieties of tobacco are in use. Valleau (23) in Kentucky reported successful use of several commercial varieties bearing the *N. glutinosa* type of resistance.

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CUCUMBER MOSAIC

A mosaic of cucurbit plants was reported in Ohio, Massachusetts, and Connecticut during the first decade of the current century, but delineation

of a distinct infectious disease was not recorded until 1916. The relatively rapid increase of the disease in many areas of the United States after the latter date brought it into the position of major importance on cucumber where it has remained up to the present. The first thorough comprehensive

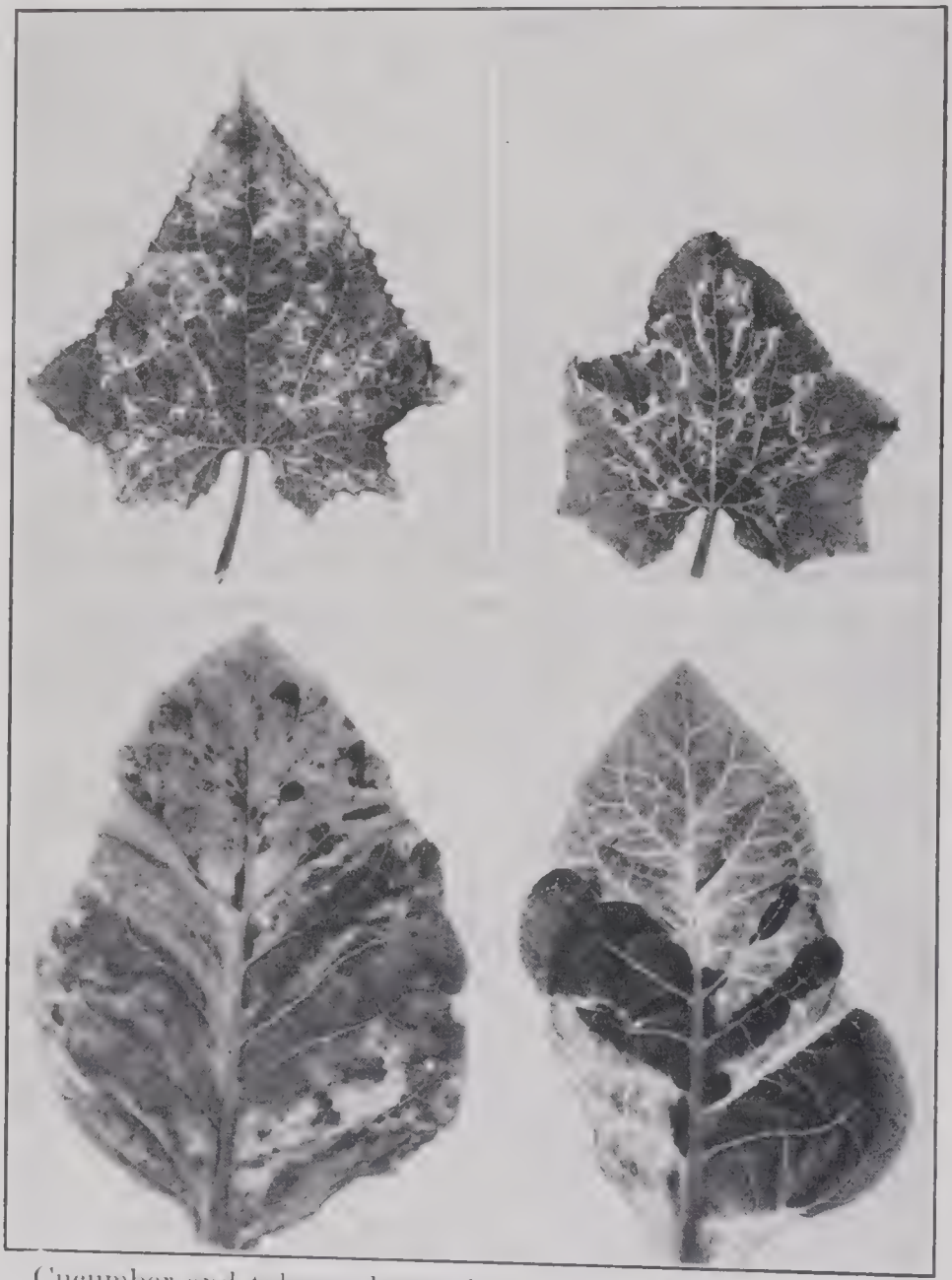

FIG. 158. Cucumber and tobacco leaves infected by the common-cucumber-mosaic virus.

study of the disease was published by Doolittle (5) in 1920. While cucurbit mosaic was at first regarded as closely confined to vine crops, the virus is now known to consist of many strains varying in host range. An extensive list of dicotyledons and some monocotyledons is now included among the suscepts of one or another strain of the virus. Among the diseases of economic crops incited by cucumber-mosaic virus, in addition to the mosaic of vine crops, are southern celery blight (9, 33, 34), spinach mosaic

(14), heart rot of banana (18), some forms of delphinium mosaic (25), pansy flower breaking (26), tomato mosaic, tobacco mosaic, string-bean mosaic (35), lima-bean mosaic (12), crucifer mosaic (22), pea mosaic (35), and beet mosaic (28). Many common weeds are suscepts. The disease is world-wide in occurrence.

There are certain other viruses which may infect cucurbits, and the resultant symptoms may be confused with those incited by the typical-cucumber-mosaic virus. Watermelon is not a host of the latter virus as a rule, but it and other cucurbits may be infected by other distinct viruses, such as cucumber viruses 3 and 4 (1) and the tobacco-ring-spot virus (21).

FIG. 159. Cucumber mosaic on cucumber fruits showing mottle and distortion. Healthy fruit at left.

Rader *et al.* (24) described a mosaic of muskmelon in New York, the virus of which is regarded as distinct from the typical-cucumber-mosaic virus. The squash mosaic of California (10, 11, 15, 19, 20) is also distinct, the virus showing some resemblance to that of tobacco ring spot.

Symptoms. The signs of infection on cucumber are often variable and difficult to define. The mosaic symptom is commonly seen on young leaves near the growing tip, but in such leaves, as they grow larger, the pattern often shifts to a diffuse, almost indistinguishable, mottle (Fig. 158). V-shaped necrotic areas may appear on older leaves, extending from the margin toward the midrib. A dwarfing of internodes of the vine becomes progressively more conspicuous as mosaic symptoms of the leaf recede.

Young fruits show symptoms ranging from a mild mottle to extensive warty malformation and often to an almost entire lack of color, a phase referred to as "white pickle" (Fig. 159). Infrequently, affected plants show a distinct wilt. After midseason, vines may show death of all leaves except for a few near the growing point. Many such plants die prematurely and of course are of little or no productive value after midseason.

The Virus.

Cucumber-mosaic virus.
Cucumber virus 1.
Cucumis virus 1 K. M. Sm.
Marmor cucumeris var. *vulgare* Holmes.

The typical-cucumber-mosaic virus is now known to have many variants, which differ chiefly in the type of symptoms on certain hosts and in host range. They are very much alike in physical properties. The thermal inactivation point is 65 to 75°C.; aging in vitro, 6 to 19 days; inactivation by dilution, 1–1,000 to 1–20,000. A strain reported by Price (23) incites only local lesions on zinnia, while other strains are systemic on that host. Price's strain is commonly used in cross-protection tests to determine whether a given strain is a member of cucumber virus 1 group. Most isolates do not affect legumes and crucifers, but strains which infect snap bean (35), lima bean (12), garden pea (35), and cruciferous crops (22) are known. Most strains incite only local lesions on cowpea, but the snap-bean strain is systemic on that plant (35). The southern celery blight described originally as celery virus 1 has been shown to be a member of the cucumber virus 1 group (9, 33, 34). It has as wide a host range as any of the isolates described, infecting many dicotyledons as well as such important monocotyledons as corn and banana.

Ainsworth (1) distinguished two closely related strains as cucumber virus 1 and cucumber virus 2; they are identical with or very closely related to the typical strain described by Doolittle (5). He also described two other viruses as cucumber virus 3 and cucumber virus 4; these are nontypical in that the properties are different and the host range restricted to cucurbits. The virus proteins of the last two viruses have been purified (2, 16, 17). The tobacco-ring-spot virus also infects cucurbits, and whereas the typical-cucumber-mosaic virus does not infect watermelon, the former virus incites a severe disease in that host (21). Squash mosaic, as defined by Freitag (10, 11), Kendrick (15), Takahashi (31), and Middleton *et al.* (19, 20) in California, resembles the ring-spot disease; the two viruses have rather similar properties, and the proteins of each appear as spherical particles under the electron microscope (30). Takahashi (31) secured squash-mosaic-virus crystals from a purified preparation of the spherical particles.

Disease Cycle. Doolittle and Walker (7) concluded that the typical-cucumber-mosaic virus was rarely transmitted, if at all, with seed of cucumber, muskmelon, squash, and pumpkin. They did find wild cucumber seed, however, a common means of overwintering of the virus.

Bewley and Corbett (3) in England considered that the virus was transmitted in seeds of hothouse varieties of cucumber. The New York muskmelon-mosaic virus described by Rader *et al.* (24) was transmitted in muskmelon seed with rather high frequency. Kendrick (15) and Middleton (19) found the squash-mosaic virus to be borne in a low percentage of seed of muskmelon and squash.

Perennial host plants are the most common between-crop reservoir of the virus. In northern states milkweed, *Physalis* spp., and pokeweed are common weed hosts. In Florida, Doolittle and Wellman (9) found the wild wandering Jew (*Commelina nudiflora* L.) the chief interseason host of the southern-celery-mosaic strain. Current-season spread is chiefly by aphids. The melon aphid and the peach aphid are both effective vectors, while both the spotted and striped cucumber beetles also transmit the virus. When cucumbers are picked frequently for pickles, mechanical transmission may become an important factor.

Control. In greenhouse culture of cucumber, screening of ventilators to exclude aphids and frequent fumigation ordinarily control the disease (6). The eradication of wild hosts was studied extensively as a possible control measure and found to be feasible by Doolittle and Walker (8). The procedure is economically sound, however, only in very special cases, *e.g.*, with celery in Florida (34).

The development of resistant varieties of cucumber is the most practical solution of control. Highly resistant varieties suitable as breeding parents are found in certain oriental varieties such as Chinese Long and Tokyo Long (29). Suitable resistant varieties are being introduced (36).

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POTATO MOSAICS

Potato mosaic began to emerge from the complex known as potato degeneration disease about 1914 when it was described by Orton (20) as a malady present both in the United States and in Europe. Beginning about 1920 it became apparent to investigators of potato viruses that more than one type of mosaic occurred. There are now known to be several distinct viruses of potato which incite mosaic symptoms. Much confusion arose in the literature, however, owing to a number of influential factors which were only gradually realized and evaluated. The important variables may be listed tersely as follows: (*a*) Each virus concerned consists of a number of strains which differ in the symptoms they incite on the varieties which they are capable of infecting. (*b*) Each host variety, being a vegetative clone, has a high degree of phenotypic uniformity but may respond differently from each other variety to one or another virus group, the reactions varying from complete immunity through symptomless carriage, systemic necrosis, and various degrees of visible symptoms. (*c*) Environmental factors, such as temperature and light, influence the expression of a given virus-host interaction over a range extending from complete masking to severe stunting, mottling, and necrosis. The final exact diagnosis of a mosaic disease, therefore, very often cannot be based on symptoms but must rest upon the physical properties of the virus extract, serological tests, cross-protection tests, and upon standardized hosts' reactions under specific environmental conditions.

Among the mosaic diseases of potato three are widespread and rank high in importance. They are designated herein as (*a*) latent mosaic; (*b*) mild mosaic, or crinkle mosaic; (*c*) rugose mosaic.

Symptoms. *Latent Mosaic.* Standard varieties of potato which had been in culture in the United States for some years previous to 1920 are symptomless carriers of the latent-mosaic virus (Fig. 160). This is true of many European varieties as well. Some varieties used in Europe, when infected, respond with an acute systemic reaction, known commonly as top necrosis, which may take the form of killing, beginning at the tip of

the main stem or of a branch and proceeding down the top until the plant is killed. Tuber production may be prevented in top-necrotic varieties, and if small tubers are formed, they commonly show internal necrosis. Such a variety is essentially a noncarrier and is, for practical purposes, resistant. Still other varieties show chronic mottle, stunting, necrotic spots on leaves, and in some cases necrotic spots in the tuber. Only these, therefore, exhibit mosaic symptoms. Some varieties show a great deal of

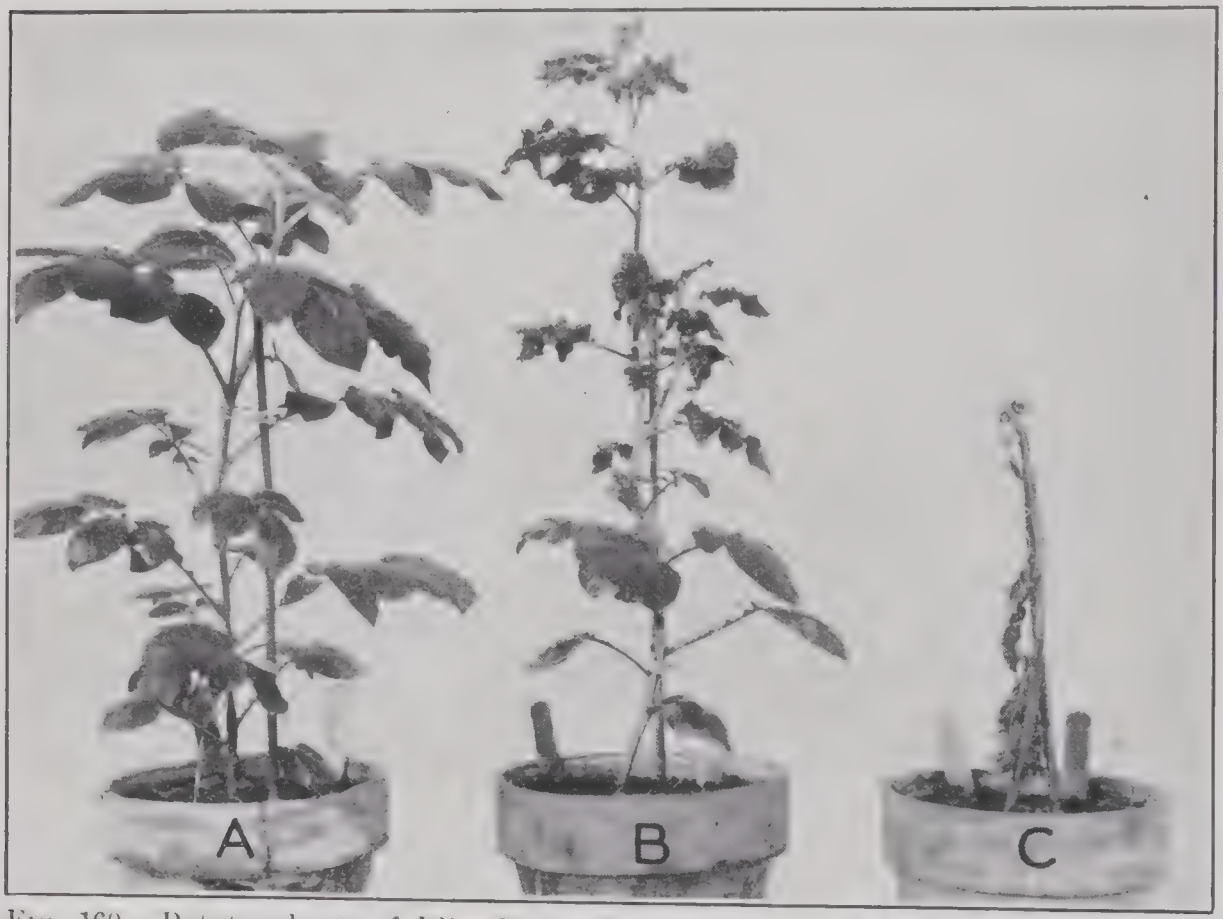

FIG. 160. Potato plants of Bliss Triumph variety. *A*, infected with the latent-mosaic virus but showing no symptoms; *B*, infected with the latent-mosaic virus and the mild-mosaic virus, showing severe mottle and stunting of leaves; *C*, infected with the latent-mosaic virus and the veinbanding virus, showing necrosis and leaf drooping characteristic of severe rugose mosaic. (*Courtesy of James Johnson.*)

variation between individuals; this is particularly true in newly introduced varieties and will be discussed more fully later.

Mild Mosaic. The disease is incited by one or another of the strains of the mild-mosaic virus, and in the case of symptomless carriers of the latent-mosaic virus the synergistic effect of the latter may be a factor in disease development. This disease has also been referred to in America as crinkle mosaic (31). Schultz and Folsom (24) at one time regarded mild mosaic and crinkle mosaic as distinct diseases. Dykstra (10) in a later comparative study considered them to be incited by related strains of

the same virus. Many old American varieties are susceptible to this disease, *e.g.*, Bliss Triumph, Green Mountain, Early Rose, Early Ohio. Others, such as Rural New Yorker and Russet Rural, though not free from symptoms, are rather highly resistant. Among European varieties are some which are symptomless carriers and some which become systemically necrotic when infected. The symptom on susceptible varieties is mottle, varying from a mild diffuse chlorosis in some to very pronounced mosaic in others (Fig. 160). Warm temperatures promote masking of symptoms; cool weather favors severe mottling and stunting. Affected plants produce undersized tubers, which usually show no visible symptom other than smallness.

Rugose Mosaic. This disease is incited by the veinbanding virus, and in the case of symptomless carriers of the latent-mosaic virus the synergistic effect of the latter is the chief factor in symptom production. Mottle is relatively inconspicuous in most cases. Rugosity and curling of the surface of the leaflet together with some stunting are the most prominent early symptoms. Necrosis usually follows, starting as individual spots on leaflets and progressing until leaflet, and eventually the entire leaf, dies, sometimes dropping but often clinging to the stem (Fig. 160). Necrosis is most severe on the lowest leaves and progresses upward slowly, a tuft of rugose leaves often persisting at the tip until the top dies prematurely. The tubers ordinarily show no symptoms other than the reduction of size induced by damage to the top.

The Viruses.

1. Latent-mosaic virus.

Potato mottle and ring-spot virus.
Potato-acronecrosis virus.
Potato virus X.
Potato virus 16.
Solanum virus 1 K. M. Sm.
Annulus dubius Holmes.

One of the first evidences of this virus was that noticed by Johnson (14) when juice extracted from apparently healthy potatoes was found to incite a mild mottle of tobacco and tomato and sometimes ring spot on tobacco. Koch (15) later separated a mottle strain and a ring-spot strain. Salaman (22) defined several strains of the virus and was the first to show that one strain, if inoculated in advance in tobacco, protected against another even more severe strain. Investigators in several countries have described strains, many of which differ in type of symptom incited and in severity of disease development. While cross-protection is rather complete on tobacco, it is not uncommon to find two or more strains in a given potato

plant. There are indications that the virus mutates frequently in the potato, and "new" strains are isolated readily when special techniques are employed.

In general the virus is relatively highly resistant to physical agents. The inactivation points are: for dilution, 1–10,000; heating, 66°C.; aging in vitro, 2 to 3 months. The virus was isolated in crystal form by Bawden and Pirie (4). The host range includes tomato, tobacco, pepper, petunia, henbane, jimson weed, and black nightshade. No insect vector has been found. The virus is transmitted readily by mechanical means.

2. Mild-mosaic virus.

Super mild-mosaic virus.
Crinkle-mosaic virus.
Potato virus A.
Solanum virus 3 K. M. Sm.
Marmor solani Holmes.

The virus is transmitted mechanically and by several species of aphids. The physical properties include thermal inactivation, 50°C.; inactivation by aging in vitro, 24 hr.; inactivation by dilution, 1–100. The host range includes tobacco and several other solanaceous species.

3. Veinbanding virus.

Potato virus Y.
Solanum virus 2 K. M. Sm.
Marmor upsilon Holmes.

This virus is now known to affect a wide host range, including black nightshade, jimson weed, tomato, henbane, petunia, and tobacco, in which it incites a distinct veinbanding symptom. The physical properties include thermal inactivation, 52°C.; inactivation by aging in vitro, 36 hr.; inactivation by dilution, 1–1,000. It is transmitted mechanically and by several species of aphid.

Disease Cycles. The chief means of perpetuation of the three potato viruses is by way of the seed tuber. Local dissemination of the latent-mosaic virus is by mechanical contact, aided in the field by man, animals, and implements, and also occurs in the preparation of seed stock by the cutting knife. Aphid transmission is the major means of spread of mild-mosaic and veinbanding viruses during the growing season.

An early observation with mild mosaic was the fact that crops of Bliss Triumph grown in the southern states in midwinter from northern certified seed often had a very high incidence of mosaic although little of the disease was seen in the seed field the previous year. Johnson (13) pointed out that air temperature had an effect upon the expression of symptoms. Tompkins

(31) studied the problem further and showed that relatively short daily exposures of infected plants to 23°C. were sufficient to mask the symptoms. This explained why the disease was often overlooked in seed fields in warm midsummer weather. Latent potato mosaic is also favored by cool temperature in symptom expression on susceptible varieties. Rugose mosaic, on the contrary, is enhanced by warm weather and suppressed by cool weather.

Varietal Resistance. Complete immunity from latent mosaic is known only in one seedling, designated as S41956. This has not shown any signs of infection by any strain of the virus tested. A considerable number of varieties are top-necrotic, and since infected individuals are eliminated, the respective variety is immune for practical purposes. Many varieties are symptomless carriers; others are mixed populations of symptomless carriers and susceptibles. In the United States all old varieties are symptomless carriers; most new varieties are mixtures of those in the latter class and susceptibles. The key to the symptomless-carrier picture in different varieties lies in the fact that all varieties derived from seedlings start out as latent-virus-free stocks and gradually the population becomes infected. Those individuals which become infected by a sufficiently mild strain of the virus become symptomless carriers, and in the course of seed selection they become the bases of clones which are perpetuated. The surviving stocks of old varieties are thus found to be infected generally with a relatively mild strain which is protective against severe strains. In new varieties which are not in the immune or the hypersensitive class, one finds some plants which have become infected by a mild protective strain and other plants which are entirely free from a latent-mosaic virus. When the plants in the latter group become infected by a severe strain, they develop acute symptoms; if they, by chance, become infected by a mild strain, initial symptoms may appear, but the next vegetative progeny of such plants are mostly plants which are symptomless carriers. According to Stevenson *et al.* (30) immunity in S41956 is controlled by two genes, *A* and *B*, both of which are necessary for expression of the character. Cadman (7) and Cockerham (9) attribute the top-necrotic reaction to a given strain of latent-mosaic virus to the control of a single gene.

While certain European varieties are known to be top-necrotic when infected by the mild-mosaic virus, most varieties which are designated as resistant are only field-resistant, since they develop typical symptoms if the virus is introduced by grafting. Among the field-resistant varieties are Katahdin, Chippewa, Sebago, and many other subsequent introductions by the U.S. Department of Agriculture. Very susceptible varieties are Bliss Triumph, Red Warba, and Green Mountain.

While seedlings differ in their reaction to rugose mosaic, none of the commercial varieties in use in the United States is highly resistant.

In the breeding programs in the United States the immunity of S41956 is being introduced into new varieties. In the British Isles the necrotic reaction character with respect to all three viruses is being used in breeding lines.

Control. When varieties susceptible to one or another of the mosaic diseases are grown, the first essential control measure is the use of seed tubers low in virus content. This is made possible through the production of certified seed (see p. 610) which has a very low incidence of infected tubers. Old varieties are generally symptomless carriers of the latent-mosaic virus, and varieties field-resistant to mild mosaic can be had for most needs.

Since there is evidence at hand from experiments in the United States (26), England (29), and Australia (1) that yield is reduced somewhat in a symptomless carrier of latent-mosaic virus when compared with virus-free clones of the same variety, attention is being given in Great Britain and in Australia to the selection and maintenance of potato stocks free from latent-mosaic virus as well as from other viruses. It remains an open question whether the gain from such a procedure will justify the cost involved.

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BEAN MOSAICS

A mosaic disease of bean was first noted in 1899 by Iwanowski in Russia. It is now known that several distinct viruses may affect the common bean (*Phaseolus vulgaris* L.). The disease incited by a seed-borne virus and now known as common mosaic received first attention. In 1934 Pierce (16) defined a second virus and named the disease it incited yellow mosaic. The two viruses have been shown to be closely related (5), and they differ

chiefly in host range and in the fact that one is seed-borne and the other is not. They are the most important mosaic diseases on the crop and often cause severe reductions in yield. They have been found throughout the United States and in many parts of the world. Curly top of bean is very severe in the United States west of the Continental Divide. It is incited by the sugar-beet curly-top virus. Southern bean mosaic and pod mottle have symptoms some of which resemble those of common and yellow mosaic, but the properties of the viruses are quite distinct (28, 31).

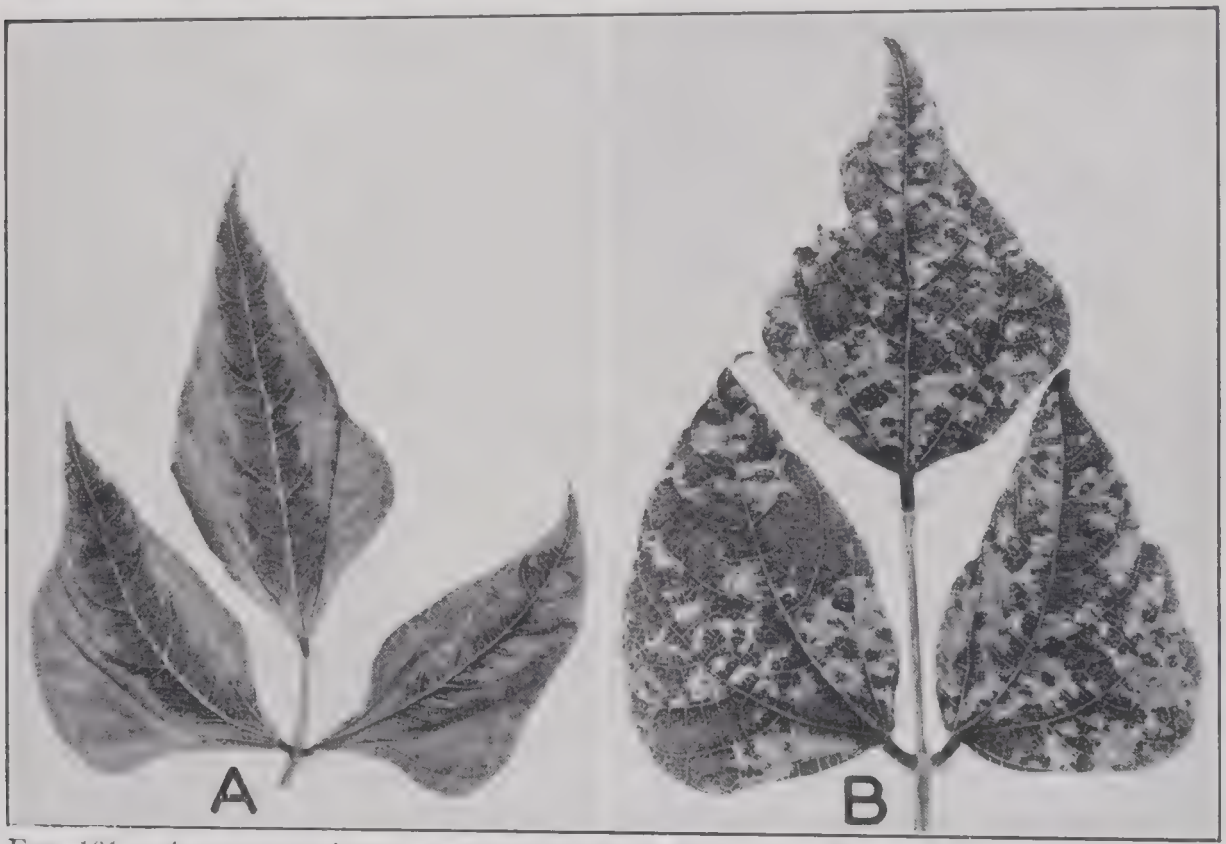

FIG. 161. *A*, common bean mosaic on leaf of Stringless Green Refugee variety; *B*, yellow bean mosaic on leaf of Idaho Refugee, which is resistant to the common-mosaic virus. (*After Pierce.*)

Symptoms. On susceptible varieties common mosaic causes various degrees of veinclearing and mottle on the leaves, the greener portions of the pattern often becoming decidedly puckered (Fig. 161). In some varieties extreme malformation of leaves may occur. Pods ordinarily show no signs, but under some conditions chlorotic spots may appear, and sometimes a glossy sheen (hydrosis) of immature pods is noticeable. The symptoms of yellow mosaic overlap those of common mosaic, and the two diseases are often difficult to distinguish in the field. In general, yellow mosaic is more severe then common mosaic, and it occurs on many varieties which are resistant to the latter. In plants affected with one strain of the yellow-mosaic virus, pods are distorted severely (6). With each disease, plant growth is reduced; blossoms tend to drop; and pods are shorter then normal.

Under exceptional circumstances the variety Corbett Refugee and varieties derived from it, which are resistant to common mosaic, show extreme necrosis of leaves, stems, and roots due to infection by that virus (Fig. 162).

FIG. 162. Bean-mosaic hypersensitive reaction of resistant Idaho Refugee to common-mosaic virus. When susceptible Stringless Green Refugee and resistant Idaho Refugee are joined by an approach graft, as in *A*, and the susceptible plant is inoculated with the common mosaic virus, the following events ensue: Typical mottle appears on leaves of the susceptible variety. Necrosis beginning at the growing tip appears in the resistant plant, as shown in *A*, and proceeds down the stem into the root, finally killing the plant. The advancing necrosis is concentrated in the phloem region as shown in *B*, where the susceptible plant is on the right and the necrotic resistant plant is on the left. If inoculations are made as pods are forming, necrosis in the resistant plant extends to the pod, as shown in *C*, where a healthy pod is shown on the right. (*After Grogan and Walker.*)

This has been shown to be a reaction which these varieties exhibit when they become systemically infected through the phloem (7, 30). The symptoms are similar to those of a virus disease described by Jenkins (10) as black root.

The Viruses.

Common mosaic: Bean virus 1.

Phaseolus virus 1 K. M. Sm.
Marmor phaseoli Holmes.

Yellow mosaic: Bean virus 2.

Phaseolus virus 2 K. M. Sm.

The properties of bean virus 1 have been determined by Fajardo (4) and Pierce (16). Reddick and Stewart (22) reported seed transmission in 1919. They pointed out in 1918 (20) and 1919 (21) that the variety Robust was highly resistant to mosaic in New York. The resistance of Robust was observed by other workers in other states. Pierce (16) noted a mosaic of Robust in Wisconsin and from infected plants recovered a virus which had similar physical properties but was not seed-borne. It affected some bean varieties and other species which had been shown to be resistant to bean virus 1. He described the new virus as bean virus 2. An important means of differentiation was based on the fact that bean virus 2 was infectious to Robust, Corbett Refugee, and Idaho Refugee and was not seed-transmitted, while with bean virus 1 the opposite was true. In 1943, Richards and Burkholder (23) described a virus in New York which resembled bean virus 1 in being seed-borne and noninfectious on Corbett Refugee and Idaho Refugee, but it was infectious on Robust and Michelite, a new variety derived from Robust. What appears to be the same virus was described by Dean and Hungerford (2) in Idaho in 1946. This virus was regarded by Grogan and Walker (7) as a strain of typical bean virus 1 and was designated by them as Burkholder's strain. Grogan and Walker (5) in 1948 described from Wisconsin a new strain of bean virus 2, which differs from the typical strain in that infection results in severe pod distortion on bean and the virus does not infect sweet clover. Zaumeyer and Thomas (29, 30) described a new virus on bean, which they called the greasy-pod, or shiny-pod, virus. Grogan and Walker (5) regarded it as a strain of bean virus 1. The appearance of various other strains of bean virus 1 and bean virus 2 is likely. That the two viruses are closely related was shown by cross-protection tests (5). The two criteria of distinction between the strains are not on the basis of symptoms, as in tobacco mosaic, but on the basis of host range and seed transmission. Some of the points of similarity and difference between two strains of each virus are given in Table 2. The differential reaction of bean varieties to the strains is of particular importance because the major approach to control of the two diseases has been through breeding for resistance. Both viruses are transmitted by aphids. Gladiolus is a host of bean virus 2.

TABLE 2. COMPARISON OF PROPERTIES AND HOST REACTIONS OF TWO STRAINS EACH OF BEAN VIRUS 1 AND BEAN VIRUS 2

Property or Host Reaction	Bean Virus 1		Bean Virus 2	
	Typical strain	Burkholder strain	Typical strain	Pod-distorting strain
Seed transmission	+	+	0	0
Aging in vitro	32 hr.	..	32 hr.	32 hr.
Thermal inactivation	58°C.	..	58°C.	60°C.
Dilution end point	1–2,000	..	1–2,000	1–2,000
Infectivity on				
Bean:				
Stringless Green Refugee	+	+	+	+
Idaho and U.S. No. 5 Refugee	0	0	+	+
Robust and Michelite	0	+	+	+
U. I. 15	0	+	+	+
U. I. 59, U. I. 81, and U. I. 123	0	0	+	0
Pea:				
Perfected Wales	0	.	+	+
Wisconsin Perfection	0	0	0	0
Broad bean	+	..	+	0
Alfalfa	0	0	0	0
White sweet clover	0	0	+	0
Lima bean	+	.	0	0
Soybean	0	..	+	0
Red clover	0	.	0	0
Alsike clover	0	..	+	+
Crimson clover	0	..	+	+
White clover	0	..	0	0

Disease Cycle. The common-mosaic virus is introduced annually with infected seed. The overwintering inoculum of bean virus 2 is found in perennial legume hosts, of which sweet clover is the commonest for the typical strain. While mechanical transmission of each virus is possible, spread in the field by this means is minor. Both viruses are aphid-transmitted, and in the case of bean virus 1 Zaumeyer and Kearns (27) have shown that a large number of species may act as vectors. Fajardo (3) and Harrison (8) showed that at temperatures below 16°C. and above 28°, the symptoms of common mosaic tend to be masked, while their fullest expression takes place between 20° and 28°. Bean virus 1 may be carried with the pollen (14). The percentage of infected seed varies with the time of infection of the plant in relation to maturity of a given pod on the plant. Generally speaking, little more than 50 per cent of seeds become infected.

Varietal Resistance. Robust (20, 21) was the first variety which showed high resistance to bean virus 1. It was developed in Michigan by Spragg from a single-plant selection in Pea bean and came into use commercially about 1916. Robust was outstanding in that plants failed to harbor the virus in any degree when inoculated. Pierce (17) developed in Idaho several strains of Great Northern field bean which were equal to Robust in resistance. Corbett Refugee was derived by Ralph Corbett in Montana from a highly resistant individual in the very susceptible variety Stringless Green Refugee. This was used as a breeding parent to develop the resistant varieties Idaho Refugee, Wisconsin Refugee (26), and U.S. No. 5 Refugee (24). Idaho Refugee and U.S. No. 5 Refugee varieties have become widely used as snap beans.

The inheritance of resistance in Robust was studied by McRostie (11) and Parker (15) and that in Corbett Refugee by Parker (15) and Pierce (18). These workers did not arrive at definite conclusions as to the nature of inheritance. Pierce (18) showed that in the F_2 progeny from a cross between Robust and Corbett Refugee, both resistant to bean virus 1, segregates susceptible to this virus appeared. This indicated separate genes for resistance in the two varieties. The genetics of resistance in these two varieties or in varieties derived from them was cleared up by Ali (1). He found resistance of Idaho Refugee and of U.S. 5 to be dominant to susceptibility of Stringless Green Refugee, while the resistance of Robust was recessive to the susceptibility of Stringless Green Refugee. Resistance and susceptibility in these three types are dependent upon the interaction of two alleles, *Aa* and *Ii*.

A is a dominant gene for susceptibility.
I is a dominant inhibitor which prevents the expression of susceptibility due to *A*.
a is the recessive gene for resistance.
AA ii is the genotype of susceptible Stringless Green Refugee.
AA II is the genotype of Idaho Refugee, U.S. 5, and Corbett Refugee, in which phenotypic expression of resistance is due to the dominant inhibitor *I*.
aa ii is the genotype of Robust, Michelite, and probably Great Northern, in which phenotypic expression of resistance is due to the recessive allele *a*.

Grogan and Walker (7) showed that although varieties such as Idaho Refugee and U.S. 5, which derived their resistance from Corbett Refugee, ordinarily did not harbor the virus when inoculated, the virus sometimes became systemic and caused severe top necrosis. While the percentage of plants which responded in this way, following the rubbing inoculation technique, was very low, a large percentage of the plants succumbed when they were inoculated through approach grafts with a susceptible variety. Varieties such as Michelite, derived from Robust, on the other hand, re-

mained perfectly healthy when inoculated by the approach-graft method. It appears, therefore, that in the Corbett type of resistance, where the dominant gene for susceptibility is present, the effect of the inhibitor gene is complete under ordinary methods of inoculation, but when more continuous exposure to the virus is provided through the approach graft, the extreme necrotic reaction follows when *A* is present. In Michelite, where *A* is absent, there appears to be no ingress of virus and no reaction.

The genetic basis for this difference between the two resistant hosts was demonstrated by Ali (1). When Robust and Idaho Refugee were crossed, he found segregation in the F_2 generation into susceptible and resistant classes, as did Pierce (18), and the ratio was that expected from a dihybrid, *i.e.*, 13 resistant to 3 susceptible. When the plants in the resistant class were inoculated with bean virus 1 by the approach-graft method, this portion of the population segregated in the ratio of 9 top-necrotic (like the Idaho Refugee parent) and 4 nonnecrotic (like the Robust parent). The various genotype classes and the expected ratio of each are as follows:

Combination	*Disease Reaction*
1 *AA II* 2 *AA Ii* 2 *Aa II* 4 *Aa Ii*	9 resistant, top-necrotic
1 *aa ii* 2 *aa Ii* 1 *aa II*	4 resistant, nonnecrotic
2 *Aa ii* 1 *AA ii*	3 susceptible, mottled

Wade and Andrus (25) secured monogenic segregation in the F_2 generation of a cross between U.S. No. 5 and Stringless Black Valentine. The latter variety apparently has the genetic composition *AA ii*, in common with Stringless Green Refugee.

Pierce (16) pointed out that many varieties which were susceptible exhibited relatively mild symptoms, although the virus increased in the tissues. He placed these in a class designated as "tolerant." There are many varieties in this class in which resistance is of substantial commercial value. All varieties tested were susceptible to typical yellow mosaic, but many showed somewhat more tolerance than others.

Control. The only feasible control for common bean mosaic is the use of resistant or tolerant varieties. Several resistant varieties of the type of the very susceptible semideterminate Stringless Green Refugee variety are now available, *e.g.*, Idaho Refugee, Sensation Refugee, and U.S. No. 5 (24, 26). In the class of bush-type snap beans are Logan and Rival. Robust and Michelite are widely used pea bean varieties. At the Uni-

versity of Idaho several resistant strains of the Great Northern field bean type have been introduced (17). It is to be noted, however, that Robust and Michelite and some of the U. I. series are not resistant to the seed-borne Burkholder strain of bean virus 1.

Yellow mosaic is more difficult to control, but since it is not seed-borne, it is not so widely destructive. Since sweet clover is the commonest source of inoculum, a certain degree of control can be attained in some areas by not planting beans near sweet clover fields and where it occurs in headlands and fence rows. Gladiolus may also be a reservoir of this virus.

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PEACH YELLOWS AND LITTLE PEACH

Peach yellows and tobacco mosaic were the first virus diseases to be studied critically. The former disease had been known in this country since 1791. The pioneer work of Erwin F. Smith (10, 11), done in the late 1880's and early 1890's (see p. 36), was a most painstaking and exhaustive investigation. Smith distinguished two diseases of peach, *i.e.*, yellows and rosette. Another virus disease, little peach, was first noted in Michigan in 1893. Kunkel (5) many years later showed that the viruses of yellows and little peach were closely related, and he concluded that they should be regarded as strains of the same virus.

Both yellows and little peach are confined to North America and are found almost exclusively in northeastern United States and southeastern Canada. The peach-yellows virus also incites diseases in almond, apricot, nectarine, and plum, and the little-peach virus incites a disease on plum. All species of *Prunus* tested are susceptible to the yellows virus or are symptomless carriers of it, while some plum varieties are symptomless carriers of the little-peach virus (9). Until control measures were developed, the two diseases were known to destroy practically entire orchards.

Symptoms. A relatively inconspicuous veinclearing is one of the early symptoms of yellows. The leaves at the tips of infected branches turn over in a sickel-shaped manner. The margins of the leaves may roll inward. Upright-growing shoots bearing small chlorotic leaves appear later, and

normally dormant buds tend to grow out (Fig. 163). Bud growth in the spring is in advance of that in the healthy tree. Fruits ripen prematurely, and the surfaces are coarsely blotched with red and purple spots which extend into the flesh, resulting in insipid flavor. In succeeding years more diseased branches appear, and the foliage becomes more generally yellow to reddish brown, and leaves are more conspicuously rolled. Trees eventually die within 1 to 5 years after the signs of the disease appear.

Little peach appears first as a distortion and a rugosity of young leaves, which tend to become smooth as they enlarge. They are greener than normal at first, later becoming yellower than healthy leaves. Short upright branches in excessive number develop. The internodes remain short, and a general stunting follows. In successive years the number of short twigs increases. Fruits remain small and become misshapen. They tend to ripen somewhat later than healthy ones. Trees die within a shorter period than those affected with yellows.

FIG. 163. Peach yellows. *(Courtesy of U.S. Department of Agriculture.)*

The Viruses.

1. Peach-yellows virus.

Peach virus 1.
Prunus virus 1 K. M. Sm.
Chlorogenus persicae var. *vulgare* Holmes.

2. Peach little-peach virus.

Peach virus 3.
Prunus virus 1A K. M. Sm.
Chlorogenus persicae var. *micropersica* Holmes.

Disease Cycle. Smith (10, 11) was the first to demonstrate that the yellows viruses were transmitted by budding and grafting, but he was unable to transmit them by mechanical means. Kunkel (5) published evidence in 1933 to show that the peach-yellows virus was transmitted to about 10 per cent of the trees exposed to the plum hopper, *Macropsis trimaculata* Fitch. The insect was found also to transmit the little-peach virus. This hopper does not move by hopping, and it seldom flies. It feeds principally on the bark of old woody branches of old trees. Its habitat covers approximately the same range as that of yellows and little peach.

When diseased buds are grafted on healthy trees, the viruses move into the latter in 8 to 14 days. If infected buds are grafted near the base of the tree, a period of a year or more may elapse before signs of transmission and infection appear, while inoculation in this manner near the top of the tree may result in a much shorter incubation period. Kunkel (8) attributed this to the fact that the viruses move downward almost exclusively. The shortest incubation period for peach yellows was 78 days. He also established the fact that the insect did not become capable of inoculating the virus until some days after it had fed upon a diseased plant. Hartzell (2) found the range of this nontransmissible period to be from 7 to 26 days. Kunkel (6) showed that plants infected with the yellows virus were immune to the little-peach virus and vice versa. On the basis of these facts he regarded them as strains of the same virus. The peach-rosette virus had no such relation to either the yellows or the little-peach virus.

Trees infected with the yellows or the little-peach virus were cured by inserting them in a tank of water at 50°C. for about 10 min. or by keeping them in an air temperature of 34.4 to 36.3° for about 25 days, the time required for a complete cure in the latter treatment increasing with the size of the tree (7).

Control. Eradication of infected trees and disposal by burning as soon as they appear are the most effective means of control. Healthy trees may be set in place of removed ones, since the virus is not soil-borne. Rigid nursery inspection and eradication in the orchard have been successful in keeping yellows and little peach from becoming major diseases. Manns (9) recommends removal of all wild plums within 1 mile of the orchard.

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ASTER YELLOWS

One of the most widespread virus diseases in the yellows group is that known as aster yellows. It acquired this name because early in this century it became conspicuous and destructive on China aster in many parts of the United States. The disease now is known in many other countries. One of the first descriptions on the aster was by R. E. Smith in 1902 in Massachusetts. An insect vector was first reported in 1924 by Kunkel (9, 10), who transmitted the virus to many species over a range of some 150 genera in 38 families. Severin and associates extended the range still further (6, 18, 20, 22, 30 32). Aside from aster and many other ornamental flowering plants, common economic hosts are carrot, celery, lettuce, endive, parsley, salsify, onion, and potato.

Symptoms. The first sign of the disease on aster is veinclearing in the young leaves (Fig. 164). Leaves become more chlorotic as they develop. The chlorosis may affect leaves unilaterally, and sectors of an entire plant may show symptoms while the remainder appears healthy. The normal color of the petals is changed, and a greenish tinge commonly appears instead. "Breaking" of flower petals has been observed in annual phlox (23). Aster leaves which are mature when symptoms appear on the plant do not change in color. At the axils of such leaves and of those which show symptoms, dormant buds are stimulated, and yellowish thin branches

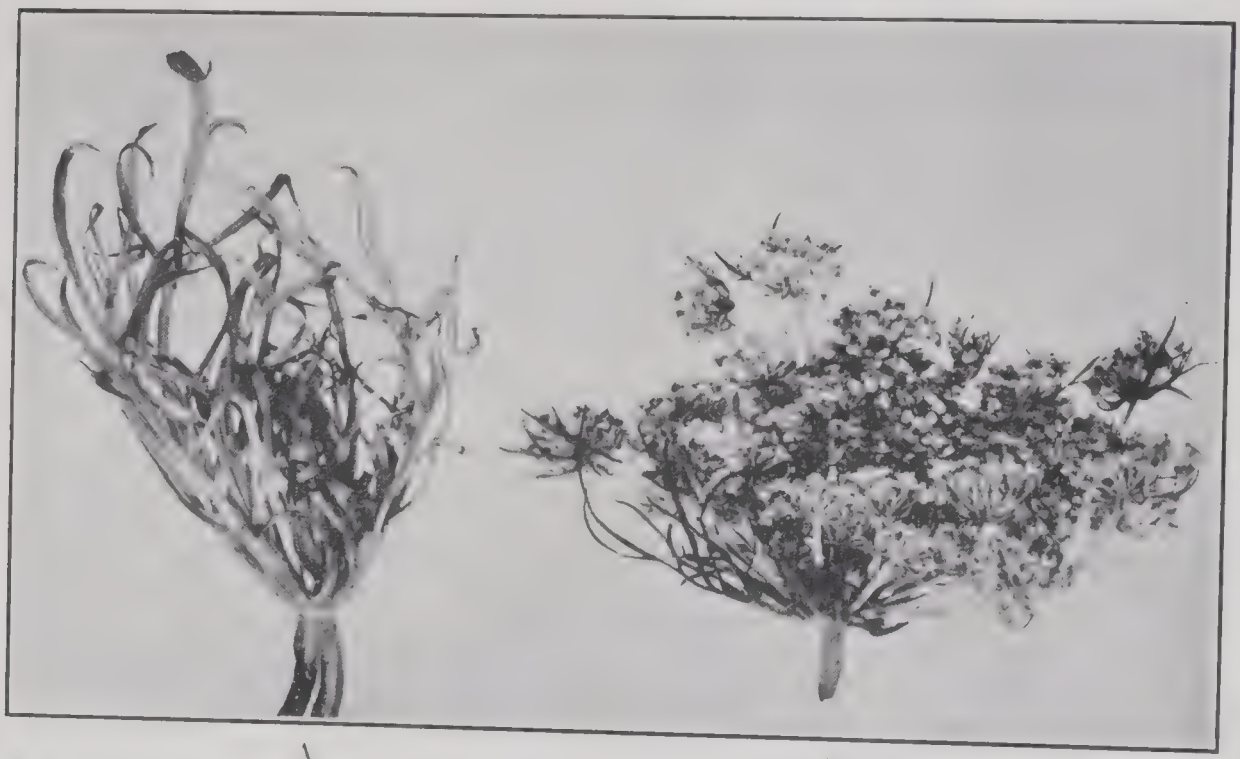
A B

FIG. 164. Aster yellows. *A*, veinclearing on young aster plant; *B*, flower distortion on onion. (*Courtesy of R. H. Larson.*)

FIG. 165. Aster yellows on carrot. Healthy plant at left. Chlorosis of leaves, proliferation of buds at the crown, and excessive production of misshapen fibrous roots on the fleshy root are the chief symptoms.

appear with internodes longer than usual. This type of proliferation is one of the striking symptoms of the disease. The main stem of the plant is stunted rather than stimulated. Leaves are more or less deformed as they grow. Flowers are misshapen and usually worthless. Necrosis of the stem a short distance below the apical bud may appear in the later stages of the disease.

The chlorosis, proliferation, malformation, and stunting occur in some degree in other plants which are affected. In lettuce the yellowing and distortion of leaves led to the name "rabbit's ear" long before the causal nature of the disease was known. The proliferating phase of the disease



A B

FIG. 166. Aster yellows on carrot seed umbels. *A*, infected plant showing distortion and sterility; *B*, healthy plant.

promotes bolting, and in this way it becomes a very destructive disease on this crop. In carrots excessive proliferation of axillary buds at the crown leads to bushy yellowish tops (Fig. 165). The quality of the root is reduced, and secondary rootlets are excessive in number and are malformed. The crowns of diseased plants are predisposed to soft-rot bacteria in moist weather. When affected carrot roots are used for seed propagation, flower umbels are distorted (Fig. 166), and the plants often die before seed is mature. The chief damage to onion is in the seed-plant stage, in which malformation and sterility of flowers occur (Fig. 164).

The purple-top disease of potato is due to infection by the aster-yellows virus. Young upper leaves become upright, and cessation of apical growth of the stem is noticeable. Leaflets do not expand normally; they roll

upward along the edges and become chlorotic. In varieties which normally contain purple pigment in stem and leaf, the leaflets take on an abnormally purple color along the edges. But proliferation and axillary tuber formation at leaf axils may follow. Many affected plants develop a permanent wilt. Necrosis occurs in the vascular system in the lower part of the stems of wilted plants and in the vascular and cortical tissues of the stem ends of tubers. In hills of affected plants, tubers are inclined to become flabby and sometimes produce very thin, weak sprouts when they germinate.

The Causal Virus.

Aster-yellows virus.
Callistephus virus 1 K. M. Sm.
Chlorogenus callistephi Holmes.

Two strains of the virus are known to occur, the chief difference being in their infectivity on celery. The strain studied by Kunkel (11) did not affect celery; that occurring in western states and studied by Severin (18) was infectious to that host. The strains are commonly referred to as the eastern, or New York, strain and the western, or California, strain, respectively.

Disease Cycle. The first insect vector to be reported was the six-spotted leaf hopper, *Macrosteles divisus* Uhl (9). More than a dozen other species of leaf hopper are now known to be vectors of the western strain (3–5, 21, 24–28).

Kunkel (9) noted that both nymph and adult leaf hoppers are unable to transmit the virus immediately after feeding upon infected plants. Although 10 days must elapse before the insects become inoculative, they are known to remain viruliferous for 100 days or longer. The virus is not transmitted mechanically from plant to plant, but Black (2) succeeded in transmitting it mechanically from insect to insect. A lapse of 11 to 45 days was required before insects to which the virus was transferred mechanically were effective in inoculating plants. Evidence was secured that a hundredfold increase of the virus had taken place in the body of the insect 6 days before the latter was able to transmit the virus to aster plants.

When colonies of infective hoppers were subjected to heat treatments of 1 day or longer at 31 to 32°C., many lost their ability to transmit the virus permanently, but some regained that ability. When they were exposed for 12 days or longer, all lost permanently their ability to transmit the virus. When hoppers recovered transmitting ability, the virus was sometimes altered so as to incite only mild symptoms on the host, a character which was retained by the virus in successive plant passages. It was suggested

that midseason decline in transmission of the virus followed by a late-season rise might be due to the reduction in virus in viruliferous insects during midsummer heat (12). This has not been demonstrated in field experiments.

When infected periwinkle plants were kept at temperatures of 38 to 42° for 2 weeks, or when they were immersed in a water bath at 40 to 45° for a few hours, the virus was completely and permanently inactivated. Such cured plants could be reinfected in the usual way, however. Infected plants of *Nicotiana rustica* L. were cured when kept at 40° for 3 weeks (13).

It is a common experience of investigators that, although the virus is transmitted to a given host species and typical symptoms follow, it is not recovered when noninfective hoppers are fed on such plants and transferred to susceptible host plants. Severin and Haasis (29) reported disease symptoms in about 50 per cent of the potato plants inoculated, but they failed to recover the virus from any of the plants. Other investigators have had similar experience with this host (14, 33). Younkin (33) and Self and Darling (17), however, recovered the aster-yellows virus from purple-top potato plants by grafting.

The virus ordinarily overwinters in northern states in perennial hosts. The time of appearance and the extent of the disease are dependent upon the insect vectors. In western New York *M. divisus* commonly hibernates on winter grain, probably in the egg stage, and possibly occasionally in the adult stage (7). The incubation period in the host plant is from 10 to 40 days or longer. It is not uncommon for the disease to be abundant in one host plant, while another potential host nearby is practically free from disease. This may be explained in part by the preference of the vector for one host as compared with another. The incidence of disease is not always correlated with the population of the vector. The relative abundance of virus reservoirs and the conditions which attract or fail to attract insects to those reservoirs are important factors in determining disease incidence.

In a study of the disease on lettuce and endive on Staten Island, New York, Linn (15) found weed barriers to be the chief reservoirs of the virus. Wind direction influenced the spread of the vector, but ordinarily the insects did not migrate much more than 200 ft. in 4 weeks.

Control. The disease is so widespread and severe on China aster in the upper Mississippi valley that commercial and home-garden culture of this plant has been largely abandoned. Commerical florists control the disease successfully by growing asters under frames completely covered with shade cloth (8). When DDT is applied to outdoor crops such as carrot at frequent enough intervals and beginning early enough in the season to reduce

hopper populations to a minimum, partial control has been accomplished (16). Linn (15) found that on Staten Island control in lettuce and endive was commercially practical by screening cold frames, transplanting to areas separated 200 ft. or more from weed borders, or destroying such weeds with sodium chlorate crystals, and dusting the transplanted beds weekly with pyrethrum-sulfur (0.15 per cent pyrethrine) or 1 per cent rotenone-sulfur. Ashdown and Watkins (1) secured reduction in the aster-yellows disease in lettuce by dusting with DDT.

In production of carrot seed in regions where aster yellows prevails, it is essential to produce the roots in locations where weed hosts do not occur or where they can be destroyed and the growing crop can be protected from insect vectors.

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CURLY TOP OF SUGAR BEETS

A serious disease of sugar beet known as western blight was recognized in California before 1900, and in that year it was found to be present in all western states where beets were grown. In 1905 the disease was associated with the presence of the beet leaf hopper (*Eutettix tenellus* Baker) and for a time was considered to be the direct effect of attack by that insect. In 1915 Bonequet and Hartung (7) reported that when leaf hoppers taken from the wild and not previously in contact with beets were allowed to feed on beets, no disease resulted. When they were next transferred to curly-top beets for from 3 to 7 days and removed thence to healthy beets, the latter contracted typical symptoms. This was the first sound evidence that the disease was due to a transmissible entity and not to the direct effect of the insect.

The curly-top disease occurs throughout the beet-growing areas west of the Continental Divide and occasionally, but not severely, in regions im-

mediately east of the Divide. It is confined to the North American continent. A similar but distinct disease occurs on sugar beet in Argentina (6). For the first three decades in the century curly top increased in destructiveness until the sugar-beet industry was seriously crippled. The development of resistant varieties initiated by Carsner (10) about 1924 has continued up to the present. Beginning about 1933 with the introduction of variety U.S. 1, resistant varieties have been introduced from time to time and have reduced greatly the damage by curly top.

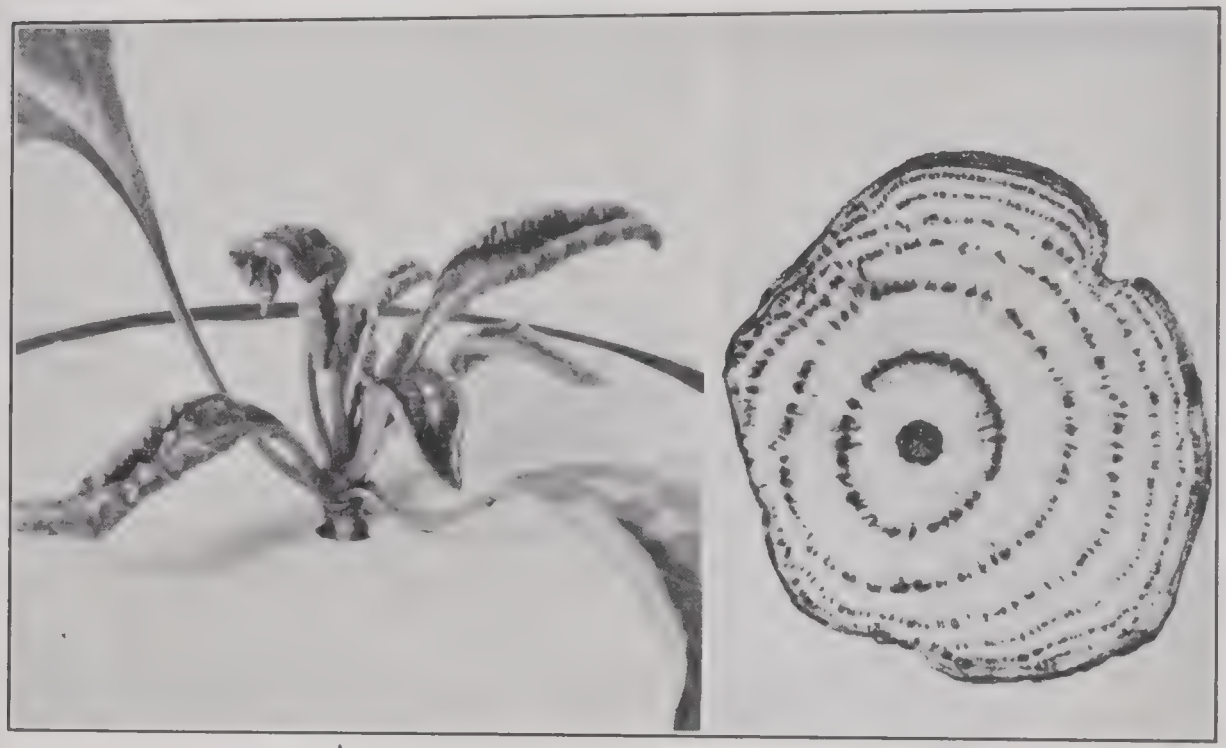
The virus has many wild and economic hosts. The former serve to provide a season-to-season reservoir. Among the economic hosts in which curly top is still a major disease are table beet, chard, spinach, tomato (30), bean (9), muskmelon, squash, and pumpkin (13). Severin (32) found 75 species included in 48 genera and 18 families to be naturally infected by the curly-top virus in California. Freitag and Severin (20) transmitted the virus experimentally to 92 species of ornamental flowering plants in 73 genera belonging to 33 families.

Symptoms. If sugar-beet plants are infected when quite young, they die rather promptly. When half-grown plants are infected, they show throughout the remainder of the season the range of symptoms by which the disease is known. The leaves which are mature when the plant becomes infected show no pathological symptoms other than to turn yellow and die eventually. The younger leaves roll inward, while the laminae tend to pucker in blister-like elevations (Fig. 167). Veinclearing appears in the youngest leaves, and this is followed by swellings on the veins on the lower side of the leaves, which assume the appearance of knot-like galls or nipple-like papillae. An exudate of liquid appears on petiole, midrib, and the lower sides of the veins. This is clear and viscid at first, becoming dark and sticky later, often drying to form a brown crust. The affected leaves may remain dull dark green, but they eventually turn yellow, then brown, and die prematurely. The environment influences the type and rate of symptom development.

Necrosis occurs in the phloem of leaf, petiole, and root, and in advanced stages of the disease dark concentric rings are visible in the flesh of the sugar beet when it is cross sectioned (Fig. 167).

In tomato, leaflets of infected plants roll upward along the midrib and become thickened and crisp as the leaf curves downward. The leaf tissue gradually turns yellow, while the veins take on a purple tinge. The plant assumes an erect habit and remains stunted. In squash young leaves have a savoyed surface or an upward roll; internodes remain short; many blossoms fail to set; and the tips of runners turn upward. Plants infected when very young are killed rapidly without the above symptoms in warm weather. Older vines decline gradually and die prematurely. In bean,

young affected plants may show killing of the growing point and yellowing of the primary leaf, followed by death of the plant. When older plants are infected, there is a downward marginal rolling of trifoliate leaflets, followed by brittleness, dwarfing, and puckering. The whole plant may become stunted and unproductive.



A B

FIG. 167. Curly top of sugar beet. *A*, symptoms on young leaves of a sugar-beet plant; *B*, cross section of a sugar-beet root showing necrosis in the tertiary phloem rings. Note that the virus has had a pronounced stunting effect in one side of the root. (*Photographs furnished by E. Carsner, U.S. Department of Agriculture.*)

The Virus.

Beet-curly-top virus.
Sugar-beet virus 1.
Beta virus 1 K. M. Sm.
Chlorogenus eutetticola Holmes.
Ruga verrucosans Carsner & Bennett.

The physical properties of the virus as determined by Severin and Freitag (35) and Bennett (2) are inactivation in vitro in extracted juice, more than 7 days; by dilution, above 1–20,000; by heating, 75 to 80°C. The virus is unusually resistant to alcohol and acetone and survives in solutions up to pH 9.1. In the phloem exudate, it withstands freezing up to 18 months. It passes commonly used porcelain filters. It remained active in thoroughly dried young sugar-beet plants for 8 years, in dried leaf hoppers for 6 months, and in dried phloem exudate for 10 months.

Disease Cycle. The virus is perpetuated between crop seasons in wild hosts (32, 33, 37), and it may overwinter in the leaf hopper (37). In semiarid regions such as southern Idaho, abandonment of land and overgrazing often tend to encourage plants which are good overwintering hosts for the insect and which may serve as reservoirs for the virus as well (17, 18). The injury to beets by transmission of the virus by the insect is dependent primarily upon the time and magnitude of spring leaf hopper movement into cultivated fields. Factors which determine the magnitude are size of fall migration to winter feeding areas, winter survival, and spring reproduction; these in turn are influenced by the type and amount of fall, winter, and spring host plants. The date of the spring migration to beet fields is influenced by the prevailing temperature. The direction of the migration is determined by prevailing winds, which are usually from the west.

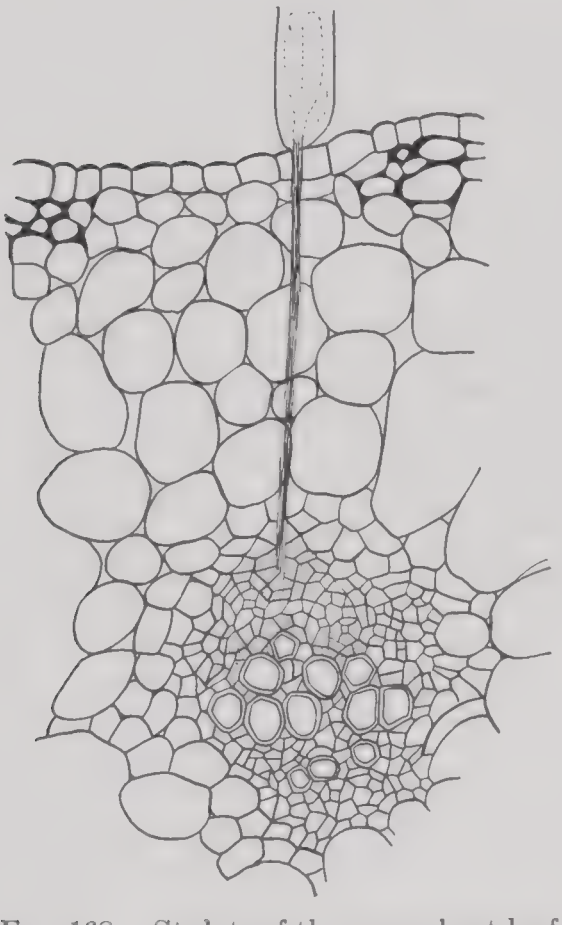


FIG. 168. Stylets of the sugar-beet leaf hopper, which transmits the curly-top virus, showing their relation to the tissues of the beet petiole during feeding. (*After Bennett.*)

The only vector known is the beet leaf hopper, *Eutettix tenellus* Baker (Fig. 168). The extract from diseased plants or infective hoppers may be used as a source of inoculum, experimentally, by allowing hoppers to feed upon it and by transferring them to the host plant (34, 35). The virus has not been transmitted mechanically. Early investigators found that hoppers were not infective until 1 to 44 days after feeding on diseased plants (11). Severin (31) secured transmission in a small percentage of cases within 20 min. Bennett and Wallace (5) were not successful in securing transmission in less than 4 hr., although the insect picked up the virus within 1 min. Freitag (19) and Bennett and Wallace (5) concluded that the virus does not multiply within the insect.

Carsner (8) secured an attenuation of the virus by passage through *Chenopodium murale* L. Lackey (24) showed that virulence could be restored by passing the attenuated virus through *Stellaria media* (L.) Cyr. Giddings (21) isolated 10 distinct strains of the virus. He also presented

evidence to show that infection was dependent upon mass action of the virus introduced (23).

The relation of the virus to the host tissue and the reaction of the latter to infection has been studied critically by Esau (14, 15). The virus in leaves apparently is contained entirely in the phloem during the time it increases and is transported. After one or more of the primary sieve tubes have differentiated, the first histological signs appear in the form of hypertrophy and death of the periderm and phloem cells adjacent to the sieve tubes (Fig. 169). In cells somewhat farther removed from the sieve tubes, hyperplasia occurs; the cells involved go through some of the changes characteristic of differentiation of sieve tubes, but normal maturation is not complete. These primary phases of hypertrophy, necrosis, and hyperplasia are followed by secondary necrosis in the abnormal sieve tubes and their companion cells. The parenchyma cells around the centers of secondary necrosis next undergo secondary hypertrophy and hyperplasia, resulting in proliferation resembling wound periderm reactions. Hypertrophy and hyperplasia are not confined to the phloem. The mesophyll cells adjacent to the phloem are stimulated to grow and divide so that they become closely packed together without intercellular spaces, while the chloroplasts show signs of degeneration. This internal change in the tissue is responsible for the macroscopical veinclearing noted as an early symptom. Further hypertrophy and hyperplasia in cortical tissue result in malformation in leaf and petiole tissue, including the formation of the characteristic protuberances. Lackey (26) demonstrated the virus in the meristematic tissue of the root tips.

Bennett (3) showed that if the virus is introduced into the distal end of a beet leaf in the dark, it often fails to move out of the leaf in a period of several days, whereas when it is inoculated in a green leaf in the light, it moves out in as short a period as 4 hr. He believed that the movement of the virus has little relation to its multiplication or to its concentration but is correlated with food transport and takes place entirely in the phloem.

Varietal Resistance. As already indicated, numerous varieties of sugar beet resistant in various degrees to curly top have been developed and released for use in various areas to which they are adapted. All such varieties are carriers of the virus and may show signs of the disease under optimum conditions. They are genuinely tolerant, however, and the amount of phloem necrosis is greatly reduced, as shown by Bennett and Esau (4), while the actual amount of virus present in the tissue is less, as shown by Giddings (22). Lackey (25) found that in a fairly resistant Great Northern bean (U.I. 81) the virus was promptly inactivated in the root tip, while it persisted in the susceptible Bountiful variety. Inheritance of resistance in bean has been studied by Schultz and Dean (29).

Control. Land and crop management and controlled grazing are important measures in reducing as much as possible the magnitude of the spring migration of leaf hopper from overwintering areas to cultivated crops. Early planting and liberal fertilization of resistant varieties of sugar beet are desirable. At present certain susceptible varieties and species must be avoided in areas where the chances of heavy infestation by viruliferous hoppers are great.

Beginning with the resistant variety of sugar beet U.S. 1, there has been developed a succession of varieties of greater agronomic value and adaptability to various areas. In southern areas, where culture comes during

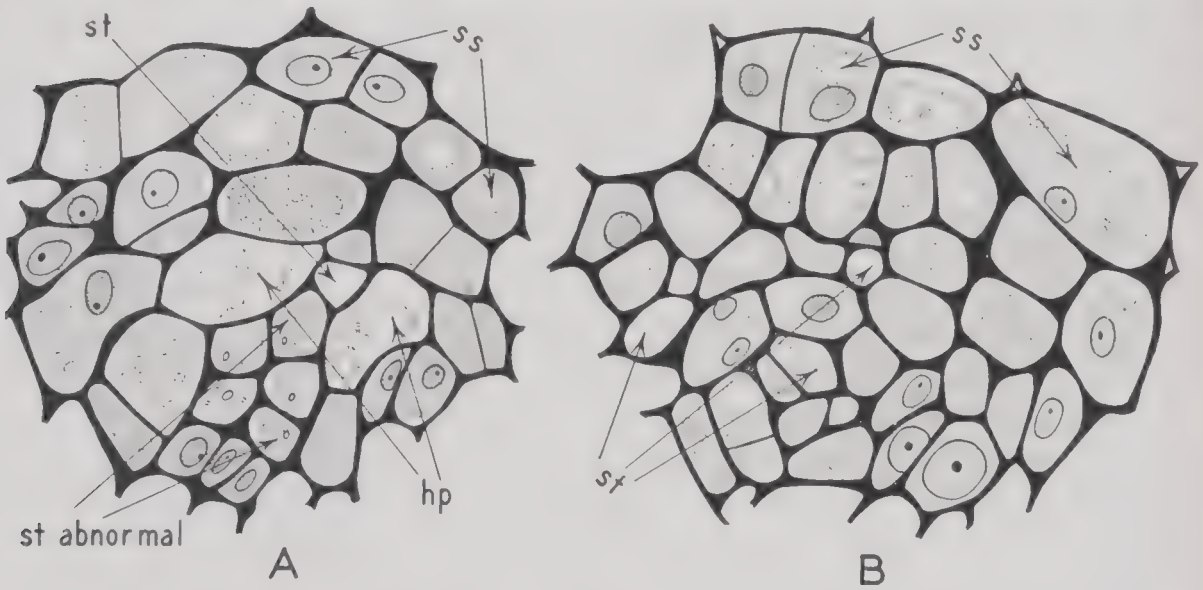


FIG. 169. Effect of the curly-top virus on the phloem of sugar-beet leaves. *A*, diseased phloem: *hp*, hypertrophied cells; *st abnormal*, abnormal sieve tubes; *st*, mature sieve tube; *ss*, starch sheath; *B*, healthy phloem: *st*, sieve tubes; *ss*, starch sheath. (*After Esau.*)

winter months, easy-bolting varieties must be avoided, although such a variety may be quite satisfactory for a summer crop in northern areas. The variety U.S. 22 occupied most of the Idaho acreage in 1942, and an improvement of this line was released in 1946 as U.S. 35. In the same year a new nonbolting curly-top and downy-mildew-resistant variety, U.S. 36, was released for use in the San Joaquin valley and in the Imperial valley of California.

Dana (13) pointed out that Umatella Marblehead and Yakima Marblehead varieties of squash are naturally highly tolerant to curly top and are of particular value in the Pacific Northwest.

In southern Idaho the Common Red Mexican bean is resistant to curly top, while the more widely grown strains of the variety Great Northern are susceptible to curly top but resistant to common mosaic. From hybrids between these two parents a new variety of the Great Northern type,

resistant to both virus diseases, has been introduced as Great Northern U.I. 15 (27). Pioneer is a curly-top resistant snap bean introduced by the U.S. Department of Agriculture and the Oregon Experiment Station.

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SPOTTED WILT

The disease known as spotted wilt was first defined as such in 1919 on tomato in South Australia, where by 1925 it had become a serious disease causing enormous losses (17). It has since been found on many crop plants and wild species in continental United States, Hawaii, Great Britain, the European continent, South Africa, Brazil, Argentina, and New Zealand. Among the economic host plants besides tomato are tobacco, potato (14, 22), pepper, lettuce, endive, celery, spinach, pea (21), and numerous ornamental species including sweet pea (20) zinnia, nasturtium, poppy, dahlia, calla lily, orchid, chrysanthemum, petunia, aster, schizanthus, salpiglossis, cineraria, and gloxinia. The yellow spot of pineapple destructive in Hawaii was shown to be incited by the spotted-wilt virus (15, 16). A list of hosts was compiled by Smith (18).

The virus concerned is distinctive in that it is transmitted by species of thrips, rather than by one of the more common groups of vectors of plant-infecting viruses.

Symptoms. Since the disease is most widely known and commonly destructive on tomato, the symptoms on that host have been studied most extensively (1, 2, 17). In all hosts, it is well to note, the symptoms may be mild under certain conditions and quite different symptomatically and more severe in another environment. On tomato one of the first characteristic signs is bronze-colored marking on the upper side of young leaflets, which may be accompanied by a slight downward curling of the leaves. The intensity of the bronzing varies, and it is undoubtedly influenced by the

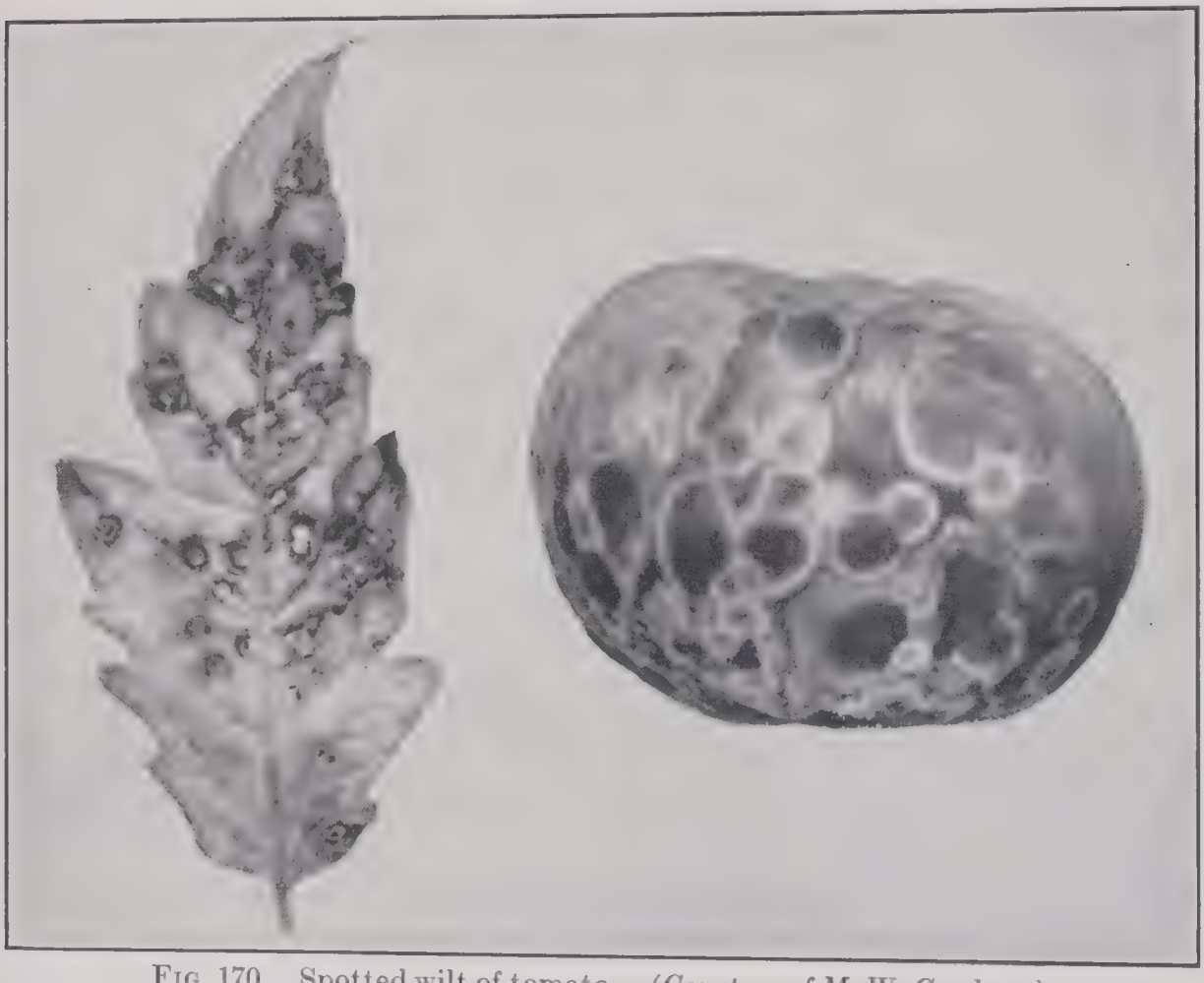

FIG. 170. Spotted wilt of tomato. (*Courtesy of M. W. Gardner.*)

environment; it may extend to petiole, stem, pedicel, and calyx. Growth may be checked permanently or only temporarily. Upward marginal rolling and stiffening of leaflets follow, and small circular necrotic spots commonly appear on the leaves under optimum conditions (Fig. 170). Necrosis extends to the stem near the growing tip, causing the latter to wilt and die. Some mottle, chlorotic spotting, and malformation may occur on leaves. On the green fruits, yellowish spots up to 1 cm. in diameter appear, often with distinct concentric zones of shades of yellow or bronze alternating with green and later with pink or red. The zoned fruit spots are probably the most characteristic sign of spotted wilt on tomato (Fig. 170).

On tobacco circular necrotic spots of various sizes appear on young leaves. A pronounced mottle may follow, often confined largely to the lower half of the older leaves, while necrosis of the leaf veins adds to the irregularity of the mottle pattern. Plants are stunted, and leaves may droop gradually and die. Leaves which are mechanically inoculated show local lesions up to 4 or 5 mm. in diameter.

Symptoms vary on the numerous host plants, and oftentimes symptoms are so variable as to be of little diagnostic value. Various types of zoned chlorotic and necrotic spots run through the symptoms syndrome.

The Virus.

Tomato-spotted-wilt virus.
Lycopersicum virus 3 K. M. Sm.
Lethum australiense Holmes.

The virus is inactivated by heating at 42 to 45°C. for 10 min.; at dilutions of 1–100,000; by aging in crude plant sap at 20°C. for 5 hr. Longevity in vitro is increased by low temperature and by addition of reducing agents to the extract, since the rapid inactivation of the virus in extracted sap is correlated with the activity of dehydrogenases in the mixture (6, 7). Insect vectors include onion thrips (*Thrips tabaci* L.), black carnation thrips (*Frankliniella insularis* Franklin), *F. occidentalis* Perg., and *F. moultoni* Hood.

Disease Cycle. The virus is perpetuated in plant hosts. In areas with severe winters reservoirs consist of plants growing in greenhouses or of outdoor perennial hosts, of which dahlia is an important one. In areas with mild winters, winter annuals are common reservoirs. With a wide host range among ornamental plants, dooryard flowers are often the source of inoculum by which commercial crops are infected. The disease is severe on tomato and other vegetable crops during the cool season in the San Francisco Bay region of California.

Although the virus may be transmitted mechanically, insect vectors are the most important means of dissemination. Only the larval stage is capable of picking up the virus from infected plants. Insects do not become infective until 5 to 9 days after feeding, and they remain infective into the adult stage.

Sunlight exerts an inhibitory effect upon the development of spotted-wilt lesions on tobacco (5).

Control. In greenhouse culture the infected plants which, though mildly affected, serve as a source of inoculum should be segregated and thrip control practiced.

In outdoor culture in areas where spotted wilt occurs regularly in severe form, some reduction of disease is obtained by elimination of overwintering

hosts, placement of fields some distance from potential reservoirs, particularly dooryards, and control of thrips by the most effective practical method.

Some lines of red-currant tomato are highly resistant to spotted wilt and have been used as parents in breeding programs in California (19) and Hawaii, where the disease is often a limiting factor in tomato production. The Pearl Harbor variety (13) introduced by the Hawaii Experiment Station is highly resistant to spotted wilt. Holmes (12) found that the resistance of Pearl Harbor did not hold up in New Jersey, while a monofactorial resistance found in an Argentine variety was effective in that area.

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CHAPTER 13

RELATION OF ENVIRONMENT TO DISEASE DEVELOPMENT

Before the acceptance of the germ theory in relation to plant disease, environment was considered to provide the major causal factors. As illustrated in Chap. 3 on nonparasitic diseases, there are many maladies in which the inciting agencies are entirely nonorganic. In the period from 1800 to 1860, when the autogenetic theory reached its peak and began to decline, the adverse effect of environal factors upon the plant was considered as the cause, while the spontaneous appearance of one or another fungus in the lesion was regarded as the effect. The proof of the pathogenicity of the wheat-bunt organism by Prévost in 1807 and confirmation of his work by the Tulasnes in 1847 and by De Bary in 1853 dispelled autogeneticism and stimulated research on parasitic diseases. For some 50 years after De Bary's entrance into the field in 1853, much more attention was paid by investigators to the naming and describing of new pathogens on the one hand, and to the working out of chemotherapeutic control measures on the other hand, than was given to the influence of environmental factors upon the development of parasitic diseases. Sorauer gave much attention to nonparasitic diseases and emphasized the importance of environal factors in predisposition of plants to parasitic organisms. Ward, late in the nineteenth century, in studies of plant rusts recorded the environal conditions of his experiments and pointed out the influence of environment upon the results he obtained. After the turn of the twentieth century, particularly after 1910, a rapid increase in the amount of attention to environment in relation to disease development was evident.

As complete an understanding as may be possible of the reaction of pathogen and host, independently and in combination, to environment is desirable. Important factors are aerial temperature, aerial humidity, oxygen relations, aerial contaminants such as injurious gases, frequency and amount of rainfall, soil temperature, soil moisture, soil reaction, and soil fertility. These are to be considered in their relation to perpetuation of the pathogen from one crop season to the next; to building up of the early inoculum; to the formation of spores, dissemination of inoculum, and primary infection; to the development of disease after infection; and to the production of secondary inoculum and of overwintering stages. When an insect vector is involved, the relation of environment to its development is equally important.

The experimental studies of disease development in relation to various environal factors have now become quite extensive. The consideration of

these factors can be taken up conveniently in three groups of diseases, which are designated as follows:

1. Storage and transit diseases. The pathogen is related to mature or nearly mature storage organs of the host which are exposed to a more or less artificial environment.
2. Diseases incited by soil-borne pathogens. The pathogen is a more or less permanent soil inhabitant, and infection occurs primarily through subterranean portions of the plant. Soil factors are thus the most influential.
3. Diseases incited by air-borne pathogens. The pathogen completes its life cycle primarily above ground and in relation to the aerial organs of the host. Aerial factors are thus the most important.

DISEASES IN STORAGE AND TRANSIT

In storage and distribution of perishable fruits and vegetables, disease is an ever-important factor. It is now generally recognized that the primary inoculum in most cases is to be found in the field on the crop at the time of harvest. Contamination during harvest and packaging may, in some cases, be the sole or the major source of inoculum.

Predisposing Factors. In the case of wound parasites, predisposition of the host by freezing or bruising at harvest or thereafter may become the major factor in initiation of disease. Other predisposing environal factors may come into play. In the case of onion neck rot the conditions which prevail as the neck of the bulb matures are important. Moist weather favors the development of inoculum of the pathogens and retards maturation of the host, prolonging the period of susceptibility of the neck of the bulb. Dry weather reduces development of inoculum and hastens the maturity of the neck tissue; after maturity infection does not ordinarily occur. Even after incipient infection, rapid drying of the neck tissue will effectively stop further disease development. In the case of some of the fungi which invade potato tubers through wounds incurred at harvest time, infection will not occur if cork-cambium activity has walled off the wounded tissue. Warm moist conditions are favorable to the organisms, but they are sufficiently more favorable to cork-cambium formation of the host to reduce infection effectively. Thus prestorage of potatoes at 21°C. or above for a period of about 2 weeks after harvest is commonly recommended (45). Curing of sweet-potato roots after harvest at 28 to 32°C. and at high relative humidity for a period of 10 days hastens periderm formation and thereby reduces infection by root-rotting fungi (21). Gladiolus bulbs wounded and cured at 29° and 97 per cent relative humidity for 10 days before storage at temperatures of 10° or below show very little infection from bulb-rotting fungi, in comparison with wounded bulbs stored without being

cured. This difference was shown to be due to rapid development erized periderm cells during curing (23).

Infection having taken place, temperature is usually the most imp environal factor, and humidity is the next in importance.

Temperature. In general, fungus and bacterial organisms which bring about decay of fruits and vegetables are reduced in growth with lowering of temperature below the optimum. When these organisms are grown on suitable media at a range of constant temperatures, an indication is secured of the response of growth rate to temperature. In many the optimum lies between 20 and 30°C., while above and below the optimum, growth rates decline. Optima and growth rates of a given species may differ on various substrates, and it is not uncommon to find that the fungus produces staling products (which retard its growth) more abundantly at one temperature than another. The true growth-temperature relation may thus be obscured.

In storage disease it is common to find that the range of temperature at which an organism grows coincides fairly closely with the range at which disease development may occur. Lauritzen (18) found the optimum for growth of the organism of black rot of carrot (*Alternaria radicina* Meier, Drechs., & Eddy) on carrot agar and for progress of the disease in storage to be 28°C. Fawcett and Barger (7) found the optima for growth of *Penicillium italicum* Sacc. and *P. digitatum* Wehmer and for decay of orange fruits to be nearly the same. At temperatures above and below the optimum, decay was retarded more rapidly than was growth on media, and retardation was greater with *P. digitatum* than with *P. italicum*. In black rot of sweet potato, *Ceratostomella fimbriata* (Ell. & Halst.) Elliot, the range for infection is 9 to 36°; the optimum for disease development is 23 to 27°; the range and the optimum for growth on agar are the same (17). The limits of growth of *Sclerotinia sclerotiorum* (Lib.) DBy. (p. 364) on agar are 0.9 to 32.5° with an optimum at 24°; infection of carrots was secured from −1 to 28° with greatest amount of decay in storage at 23° (20); infection of snap beans after harvest occurred from 0 to 28°, and optimum infection at 19 to 24° (22).

In the case of *Rhizopus* soft rot of sweet potato (*R. nigricans* Ehr.) the optimum for decay does not coincide with that of growth of the pathogen. The cardinal temperatures for growth are minimum, 6°C.; optimum, 23 to 26°; maximum, about 31°. Those for development of the disease in storage are minimum, 3 to 7°; optimum, 15.5 to 23°; maximum, 29 to 34°. The formation of wound cork in the host, which is a deciding factor in infection, increases with temperature up to 33°. Thus as growth of the pathogen increases with temperature, the protective action of the host also increases.

The optimum temperature for decay is, therefore, determined by the interaction of these two factors and is found to lie at a point where host reaction is relatively slow and where the suboptimum growth of the pathogen is great enough to bring about disease development. At the optimum temperature for growth of the pathogen, host reaction is near its optimum and is predominant in excluding infection.

Moisture. Moisture has varied effects upon disease development in storage. Some of the most critical studies have been made with sweet potato with reference to *Rhizopus* soft rot. At a favorable temperature (23°C.) optimum infection occurred at 75 to 84 per cent relative humidity. At 93 to 99 per cent relative humidity for 4 to 12 days few infections occurred either during that period or when the roots were removed to a more favorable humidity. A large portion of roots exposed at 51 to 73 per cent relative humidity for 4 to 12 days and then removed to the optimum humidity developed decay. The difference between the high- and low-humidity lots was attributed to the fact that at the high humidity, resistance to infection developed promptly in the form of suberized periderm formation. The practice of curing sweet potatoes at 28 to 32°C. and high relative humidity for 10 days is based on the fact that suberin and periderm formation are effective against *Rhizopus* and against other rotting pathogens such as *Fusarium oxysporum* Schlecht. (*Fusarium* end rot), *Diplodia tubericola* (Ell. & Ev.) Taub. (Java black rot), and *Macrophomina phaseoli* (Maub.) Ashby (*Sclerotium bataticola* Taub.) (charcoal rot). Since sweet potatoes develop a nonparasitic internal necrosis at temperatures below 10°C., it is necessary to store after curing at 10 to 15°C. (21).

Critical studies of the effect of relative humidity in storage on infection by carrot-rotting fungi have been made. Infection by *Sclerotinia sclerotiorum* (Lib.) DBy. (watery soft rot) is favored by 95 per cent relative humidity. Roots contaminated on the surface with the fungus showed little infection at low temperature if the relative humidity was kept down to 85 to 90 per cent. High relative humidity was also essential to bacterial soft rot. Since carrots shrivel badly at lower relative humidity, it is essential to keep the latter at 90 to 95 per cent in storage, and control of storage rots by reducing humidity is not practical. However carrots store well at 0°C., and therefore low temperature is relied upon to reduce storage decays.

DISEASES ASSOCIATED WITH SOIL-BORNE PATHOGENS

The first comprehensive study of soil factors in relation to development of diseases incited by soil-borne organisms was that initiated about 1916 by L. R. Jones at the University of Wisconsin. Primary attention was given to soil temperature, and for this type of experimental approach a thermostatically controlled water bath was devised into which watertight vessels

containing plants growing in soil or other substrate could be inserted. This apparatus is known as the Wisconsin soil-temperature tank (see Fig. 193). Much of the work reported previous to 1926 is summarized by Jones *et al.* (13).

Temperature. One group of pathogens which has received much study is the vascular fusaria. Common diseases concerned are cotton wilt, tomato wilt, flax wilt, watermelon wilt, muskmelon wilt, pea wilt, and cabbage yellows. The pathogens all subsist in the soil, enter the respective host plant through the fibrous root system, and bring about their pathogenetic influence on the host through their growth activity in the larger xylem elements. Generally these diseases are most severe in warm weather. Some are restricted in distribution by prevailing temperature in growing seasons. Tomato wilt is seldom found out of doors north of the northern border of Illinois. Pea wilt, however, extends throughout the northern states. Cabbage yellows is more severe in southern Wisconsin and regions to the south than in areas farther north. In general the growth rates of these pathogens increase with temperature to an optimum at about 27°C., above which the rate drops quite abruptly. When the diseases which the respective species incite are studied in plants exposed to various soil and air temperatures, two facts are generally true: (*a*) The soil temperature is very influential in determining the rate and extent of disease development, while air temperature has relatively secondary importance. (*b*) The disease-temperature curve often follows very closely the pathogen-temperature curve, but there are important exceptions. The various hosts involved in this group include some with relatively low temperature optima, such as pea and cabbage, and others with high temperature optima, such as tomato, watermelon, muskmelon, and cotton.

Those diseases discussed by Jones *et al.* (13) were cabbage yellows, flax wilt, and tomato wilt. In each of these there was a critical minimum temperature below which the host grew but disease did not develop. In cabbage yellows this point was about 17°, but Blank (1) has since shown that some disease may occur at as low as 12°. Above 17° the temperature-disease curve rose with the fungus-growth curve to the peak of the latter at about 28°, beyond which point both curves descended. Since cabbage grows best at about 20° and fairly well below 17°, it was possible to restrict the disease at the low temperature. Cabbage grows poorly at 28° and above, but it was possible to exclude the disease at 38° (Fig. 171). Flax wilt failed to develop at 12° and at 38° but rose in severity from 16 to 24 to 28° and descended at 31° and more so at 34°. In tomato wilt a similar relation occurred, but an important difference was that, although the tomato-wilt organism had approximately the same temperature range and optimum as those of cabbage yellows and flax wilt, the usual lower limit of

the tomato disease was about 20° and the upper limit was slightly above 33°. This led to the question, not answered at the time, whether the low and high temperature affected the tomato plant so as to make it more resistant than it was at the temperature for optimum growth of the fungus. The optimum temperatures in this case for host, pathogen, and disease are all at 28°. As a matter of fact Foster and Walker (11) later produced wilt in soil as low as 12°. Further discussion of this case will be taken up later

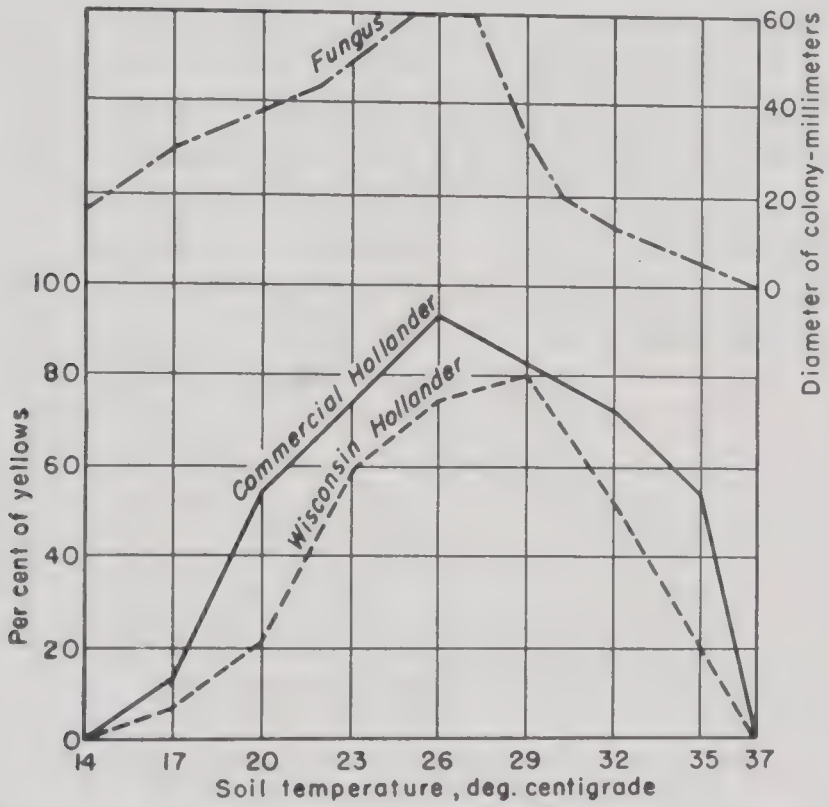


FIG. 171. Relation of temperature to the rate of growth on agar of the cabbage-yellows organism (*Fusarium oxysporum* f. *conglutinans*), compared with the development of the disease in cabbage seedlings grown in infested soil at a range of constant soil temperatures. Commercial Hollander is a very susceptible variety; Wisconsin Hollander is moderately resistant in the field but much less so in the seedling stage under greenhouse conditions. Note that the optima for fungus growth and disease development in the susceptible variety are similar (compare with Figs. 172 and 173). (*After W. B. Tisdale.*)

in the section on Predisposition. It is sufficient to say here that unquestionably soil temperature and other factors influence the resistance or susceptibility of tomatoes to wilt.

The relation of soil temperature to pea wilt was first studied by Linford (25), who in 1928 showed that, although the growth range and optimum (28°) of the pea-wilt *Fusarium* were similar to those of cabbage yellows, flax wilt, and tomato wilt, the disease curve did not follow. It rose from a minimum of about 15° to an optimum of 21°, descending to a maximum slightly above 30°. Over the range of 18 to 27° the disease eventually

affected nearly 100 per cent of the plants, but it was much slower at 18° than at 21° and still slower at 27° (near optimum for the fungus) than at 18°. In a later study Schroeder and Walker (30) studied pea wilt in sand culture, where the influence of temperature on the competition of the soil flora with the pathogen was removed. They found that under these conditions the disease curve rose to an optimum at 28° and thus fell in line with that observed for cabbage yellows, flax wilt, and tomato wilt. They observed further that at 28° the disease took on a cortical phase not found in marked degree at 21°, while in a parallel series in soil the wilt disease developed again most severely at 21°. In sand the vascular invasion was most advanced at 24°, next at 21°, and still less at 28°. These studies with pea wilt serve to illustrate the complex nature of the interaction of host and parasite and the influence of soil temperature upon this reaction. While in some instances with the vascular fusaria it may appear as though the influence of soil temperature upon disease development is expressed primarily through its effect upon the pathogen, this is not usually the case. In cotton wilt the disease optimum is slightly above the optimum for the fungus. Since cotton grows best in warm soil, it may escape the disease effectively at high temperatures, while the disease will prevail at temperatures as low as those cotton commonly encounters (48). In muskmelon wilt, as studied by Leach and Currence (24), the fungus has a typical temperature range with an optimum at 27°. The host has an optimum at about 35°; at 20° seeds germinate poorly. The disease is more severe at 19 to 28° and is decidedly less above 30°, while at 35° it tends to exhibit a necrotic cortical phase rather than a vascular wilt. The relation of temperature to watermelon wilt is essentially the same as in muskmelon wilt (43). In these cucurbit crops the greatest disease damage, then, may be below 27°, where the retarding effect of temperature upon growth of the host apparently increases susceptibility to a greater degree than it reduces pathogenetic activity of the parasite.

In the case of fusaria which are not primarily vascular but mostly cortical in their pathogenic relations to the host, the influence of environment (temperature) on the host is sometimes more pronounced. In the case of the fusarial stage of *Gibberella zeae* (Schwabe) Petch (p. 342), seedling blight is incited on both wheat, a low-temperature plant, and corn, a high-temperature plant. The growth curve for the fungus is similar to that of vascular fusaria. With both hosts the fungus is least pathogenic at the optimum temperature for the host. Thus with wheat the optimum disease development is at 20° or above, while with corn it is below 20°. When the fungus is the same in each disease, as in this case, the influence of temperature on the disease through its effect upon host metabolism cannot be questioned (Fig. 172).

Another example which has received considerable study is the relation of *Thielaviopsis basicola* (Berk.) Ferr. to the tobacco plant, in which it incites a cortical root rot. Here the temperature-parasite curve is not unlike *Fusarium* with an optimum at around 28 to 30°C. The tobacco does best at high temperatures, the highest dry weight per plant being secured at a soil temperature of 28 to 29°C. The disease is most severe at 17 to 23°, while at 29 to 30°, the period most optimum for parasite and host, there is practically no injury. It is thus evident that here again the disease is least severe when the host is most active, even though the parasite is also pre-

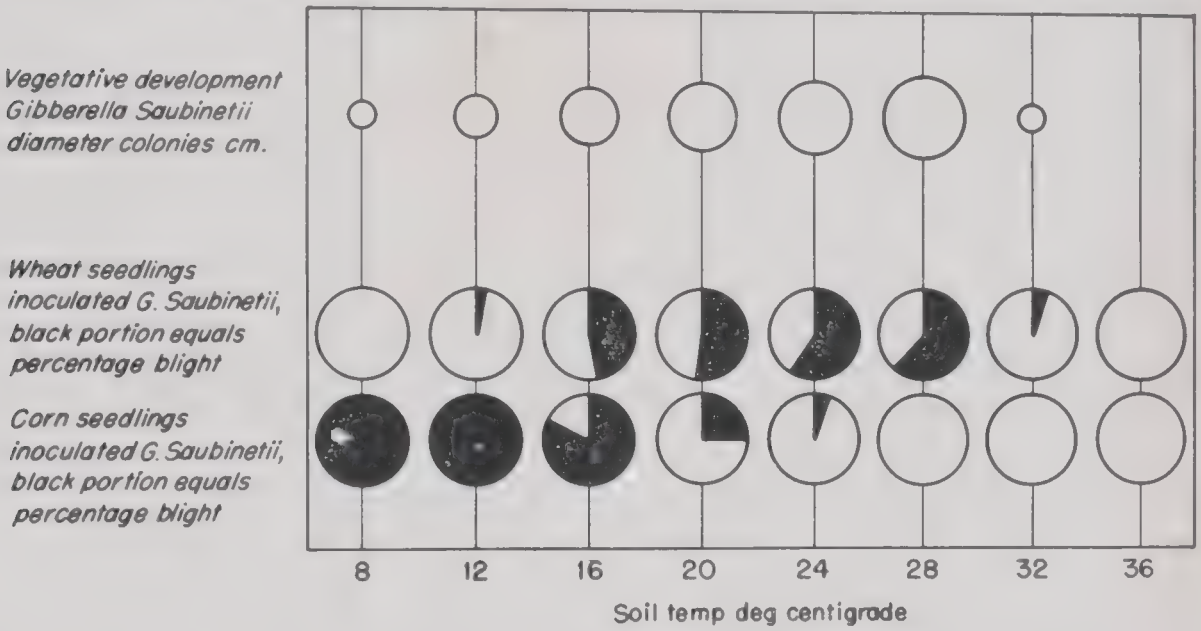


FIG. 172. A graphic summary of the development of *Gibberella zeae* (*saubinetii*) on agar at various temperatures and of seedling blights of corn and wheat growing in infested soil at a range of constant soil temperatures. The black portion of the circle in the two lowest rows shows the average percentage of diseased seedlings. Note that in the case of wheat the optimum for disease was close to that for fungus growth, while in the case of corn the optimum was some 16 to 20° lower (compare with Figs. 171 and 173). (*After Dickson.*)

sumably most active at that temperature. The nature of the pathogenic relation, however, is quite different from that in the case of vascular fusaria. The tobacco-root-rot organism invades the cortex particularly at the points of rupture of secondary roots. In actively growing roots, development of the cork cambium, as well as that of the periderm from the pericycle, is most active. These meristematic tissues tend to ward off the pathogen. At high temperatures the tobacco root cells are normally more active, and the meristematic activity is normally greater. If invasion stimulates these secondary meristems, they certainly are more responsive at higher soil temperatures. At temperatures of 17 to 20° root growth is slow, but the fungus is active enough to cause great damage to the less active root tissue.

Another example will show the effect of temperature as influenced by the

substrate. *Rhizoctonia solani*, the imperfect stage of *Pellicularia filamentosa* (Pat.) Rogers, attacks many hosts (p. 436). Moreover it does so typically as a subterranean fungus on roots and below-ground shoots, causing damping-off of seedlings, killing of potato sprouts, and canker of somewhat older stems. The fungus grows best on agar at 23 to 27°C., the temperature curve dropping off abruptly above that range. The disease as it occurs on potato, bean, and pea stems, however, is typically a low-temperature disease, the optima falling between 15 and 20° and the disease being very slight at 24 to 27° (Fig. 173). In this regard the fungus as it re-

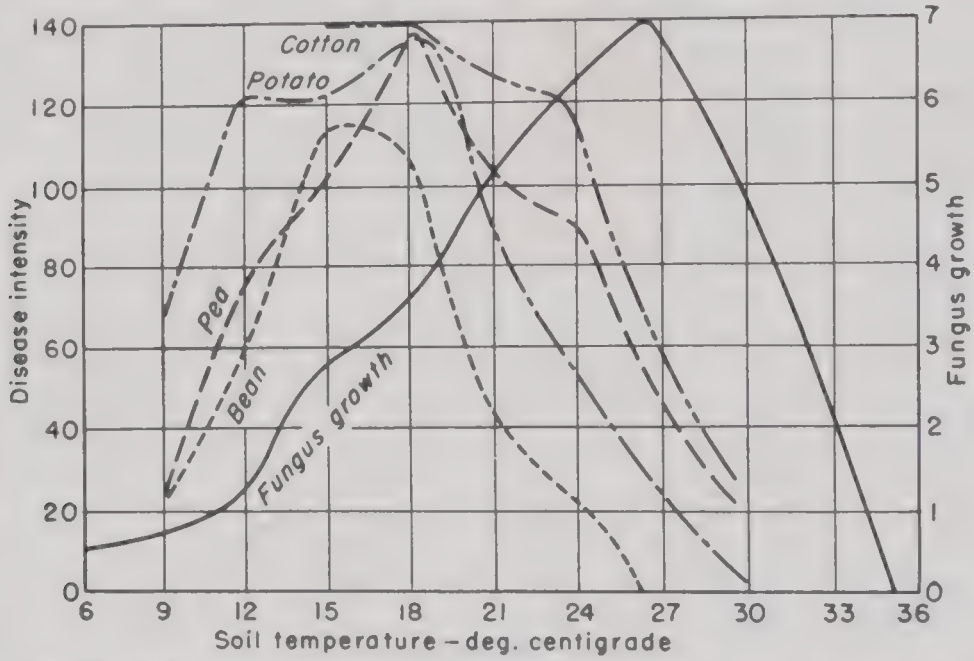


FIG. 173. Comparison of the relation of temperature to growth of *Rhizoctonia solani* (*Pellicularia filamentosa*) on agar and the development of the *Rhizoctonia* disease on stems of several hosts growing in infested soil at a range of constant soil temperatures. Note that the optimum for disease development in each case is several degrees lower than that for fungus growth (compare with Figs. 171 and 172). (*After Jones, Johnson, and Dickson.*)

lates itself to the growing host tissue is similar to *Thielaviopsis* in its temperature responses. This fungus also is a decay-producing organism on storage organs such as cabbage heads, turnip roots, and bean pods. Here it is associated with relatively dormant living tissue. In this case the optimum follows more closely that of the fungus. The optimum for head rot of cabbage is 23 to 27° (46); for rotting of turnip roots, 25° (19); for decay of snap beans after harvest, 24 to 32° (22).

Relatively few viruses are soil-borne. Yellow dwarf of potato is due to a tuber-borne virus which is affected greatly by soil temperature at the time of tuber sprouting. This disease is sometimes manifested by very poor stands. Walker and Larson (37) investigated the relation of controlled soil temperature to emergence and subsequent disease development. At 28° there was practically no emergence; there was a progressive increase in

stand at 24°, 20°, and 16°. At the last temperature nearly 100 per cent of the tubers produced sprouts above ground (Fig. 174). Low temperature not only favored emergence, but it retarded development of symptoms of the disease in the aerial parts of the plant. In the field in Wisconsin, poor stands were most pronounced when diseased tuber stocks were planted in light sandy soil in June and when high temperature prevailed for a period of about 2 weeks following planting.

In most of the diseases considered above, the host is susceptible to the pathogen over a relatively long period. The enhancement or suppression of the disease follows more or less the seasonal variation in soil temperature. There are numerous diseases, however, in which the period of susceptibility of the host is a relatively short one. The predominant effect of environal factors may be restricted to that period. Some of the best examples with soil-borne pathogens are found in the smuts, while in a later section on diseases incited by air-borne pathogens apple scab and cherry leaf spot are good examples.

Some smut pathogens are soil-borne, and some are borne as contaminants on the surface of the seed. In either case the period of host susceptibility is a relatively short one following germination of the seed, and the soil factors during early growth of the host are the important ones. In the case of onion smut, the pathogen persists in the soil for long periods. As the onion seed germinates, the cotyledon develops first and emerges above ground as the first green organ. True leaves originate below ground at the stem plate, and they grow within the cotyledon or the immediately older true leaf until they are above the ground level, when they emerge from the protecting organ. Subterranean infection must take place, therefore, in the cotyledon, which is susceptible while it is elongating but immune when it reaches maturity. It eventually sloughs, but by that time the first true leaf has passed its susceptible period and protects the next leaf. Therefore the critical period for infection is that during which the cotyledon is developing. It was shown that onion seedlings increase in rate of top growth with soil temperature up to about 25°C., and the length of the period of susceptibility of the cotyledon is reduced concomitantly. Infection occurs equally well from as low as 10°C., which is nearly the minimum for germination and growth of onion, up to 25°. Above 25° rapid reduction in infection occurs, while at 29° and above the onion seedling will grow free from infection even though in infested soil. The optima for germination of chlamydospores, germination of secondary spores (hyphal fragments), and growth of mycelium lie between 13° and 22°. Above 25° germination and growth are meager. Onion smut occurs throughout the northern United States, but it does not occur in the southern United States. There are important onion-producing sections in each. The difference between the two regions, from the standpoint of

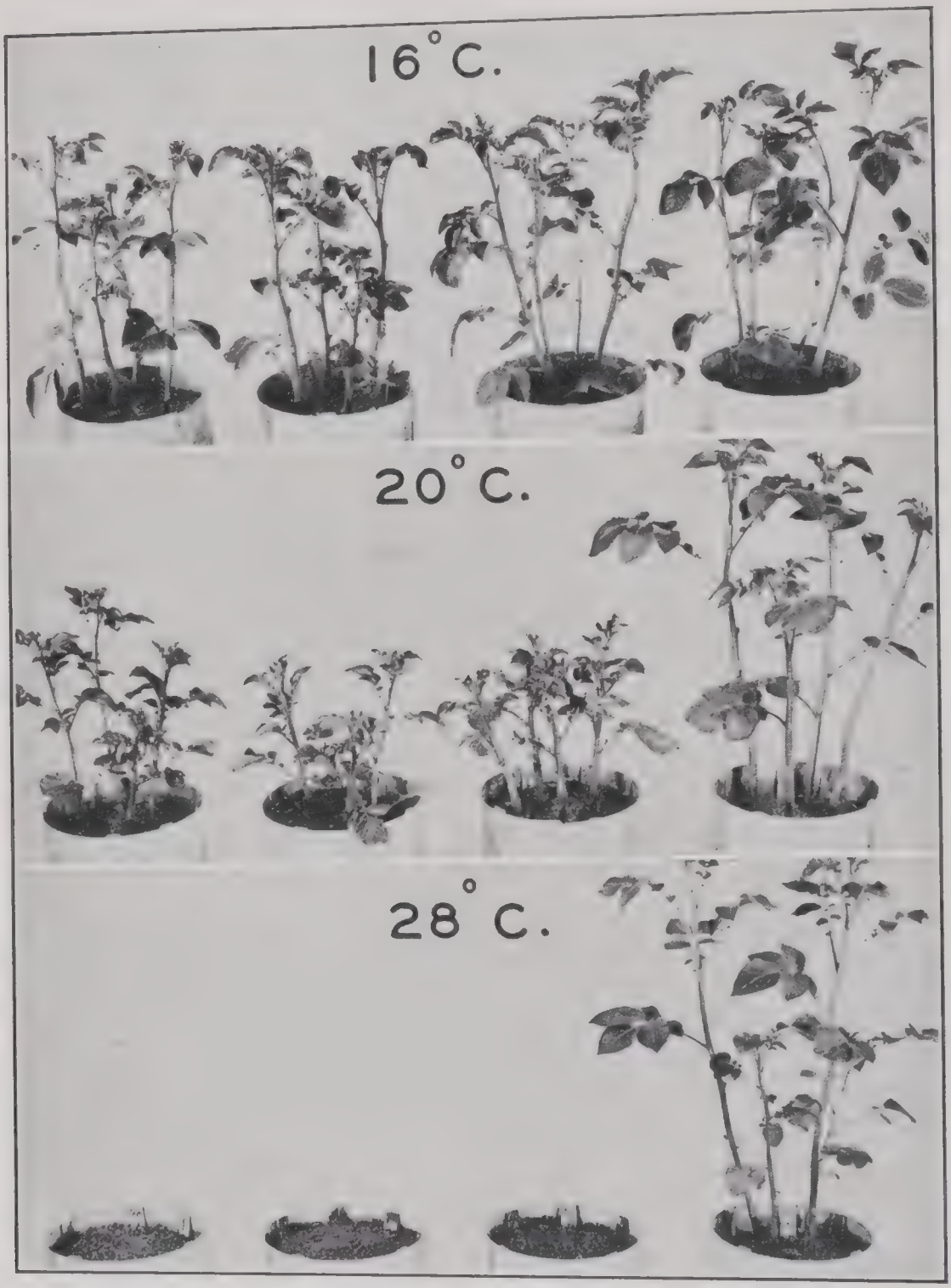


FIG. 174. The relation of soil temperature to the development of yellow dwarf of potato. Each seed tuber was divided into three seed pieces, one piece being planted in each of the three soil temperatures indicated. The can on the right in each case contains plants from noninfected tubers; the remaining cans contained infected seed pieces. Note that at 16° constant soil temperature a full stand occurred from the infected seed pieces, and symptoms were almost completely masked. At 20° nearly a full stand resulted from infected seed pieces, but growth was suppressed, and symptoms developed. At 28° infected seed pieces either did not germinate, or the shoots died before they emerged. (*After Walker and Larson.*)

the disease, is in the environment. In the North, seed is sown in the spring in cool soil when the environment is favorable to the pathogen and tends to prolong the susceptible period of the host. In the South, seed is sown in the autumn when the soil temperature is at or near its highest point of the year. At this time it is unfavorable and usually inhibitive to the fungus and tends to shorten the susceptible period of the host.

In the case of wheat bunt, a similar relation holds. Some wheat-growing areas are relatively free from bunt; in others it is a major disease. In the Palouse area of the Pacific Northwest, where autumn-sown wheat prevails, it is found that early sowings usually have less infection than late sowing. This is in correlation with the fact that the optimum for germination of winter wheat is about 25°C.; that for germination of chlamydospores of the bunt fungus is 16 to 18°; and that for infection is below 10°C. Thus in early sowing, when the soil temperature is higher than in later sowings, conditions favor shortening of the susceptible period of the host and are suboptimum for the pathogen. In other regions early spring sowings tend to have more bunt than late spring sowings. Many other investigations on temperature relations of the cereal smuts have been reviewed by Tapke (32).

Soil Moisture. In experimental studies, soil moisture is not so easily controlled as soil temperature. Since different soil types retain different amounts of water in relation to the total weight of a given volume of soil, soil moisture in a sample is given as the percentage of the water-holding capacity.

The vascular fusarial diseases, which are markedly influenced by soil temperature, are not greatly affected by soil moisture. Linford (25) found pea wilt to develop slightly more rapidly in soil at 90 per cent than at 40 per cent of the water-holding capacity. Some diseases, however, are very responsive to soil moisture. One of these is the *Aphanomyces* root rot of garden pea. Root rot is influenced by temperature, the range extending from some point below 16° to above 32° with an optimum at 24°. The rate and severity of disease development increase with the soil-moisture level up to or close to saturation. Experimental evidence under controlled conditions in the greenhouse has been corroborated by field occurrence. Another disease which is influenced greatly by soil moisture is common scab of potato. Here the pathogen is influenced in the opposite direction, the severity of disease increasing with decrease in the soil-moisture level. The initial infection of crucifer roots by the clubroot pathogen is favored by relatively high soil moisture. After infection takes place—and this may require less than 24 hr.—the influence of soil moisture is little or nil. White rot of onion is suppressed by high soil moisture and enhanced by low soil moisture (Fig. 175).

Big vein of lettuce is incited by a virus which persists in the soil and apparently infects the plant through the root system. Pryor (29) secured about 85 per cent infected plants in soil held at 85 per cent of water-holding capacity with percentages decreasing with lowering of soil moisture to about 10 per cent in soil at 35 per cent water-holding capacity.

As in the case of soil temperature, soil moisture may influence infection by smuts through its direct effect upon the pathogen or through its influence upon the length of the susceptible period of the host. The literature has been reviewed thoroughly by Tapke (32). The pathogen of flag smut of

FIG. 175. The relation of soil moisture to the development of white rot of onion (*Sclerotium cepivorum*). Onion sets were planted in infested soil held at various soil moistures. Beginning at the left the cans contain soil held at 20, 40, 60, and 80 per cent of the water-holding capacity, respectively.

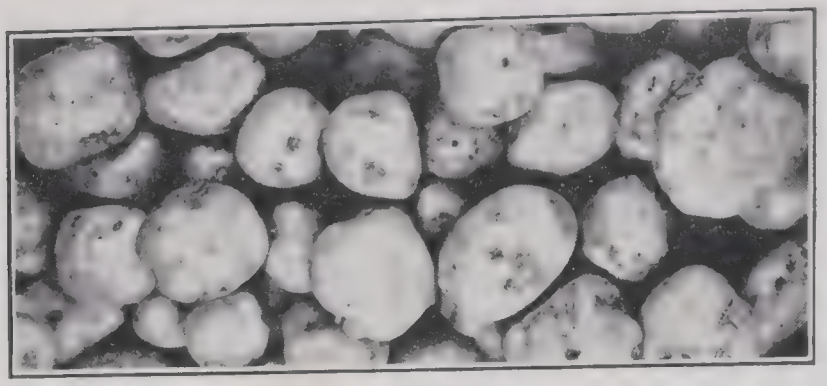
wheat (*Urocystis tritici* Koem.) is soil-borne as well as seed-borne. If wheat is sown after a dry summer and sowing is followed by a rain, spores and seeds germinate together, and infection is high. However, if rains precede sowing, infection is relatively lower, presumably because a considerable percentage of the spores germinated ahead of the seed.

Soil Reaction. The hydrogen-ion concentration of the soil solution has an effect upon the natural macro- and microflora, and it may be expected to influence soil-borne plant pathogens as well. It is surprising, however, that so few cases exist in which the influence is a striking one. Fusaria are tolerant of a wide range of soil reaction in culture and are not conspicuously limited by soil reaction, except for cotton wilt, which does not occur in any large amount on the alkaline soils of Texas. On the contrary cotton root

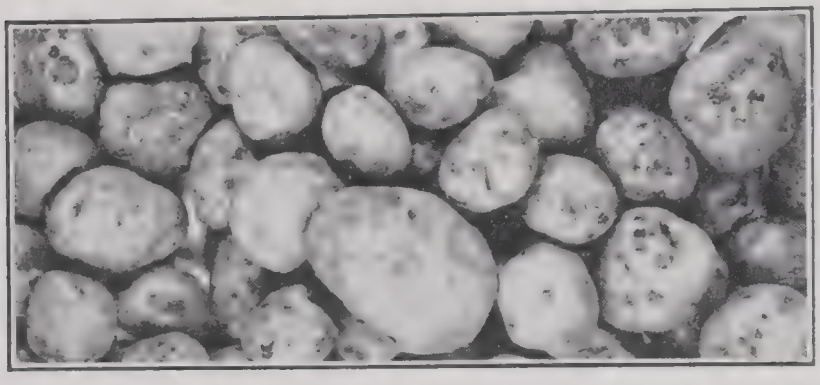
rot, *Phymatotrichum omnivorum* (Shear) Dugg., is of little importance on acid soils but is destructive on neutral and slightly alkaline soils (33). Two other important disease pathogens which show striking and opposite responses to soil reaction are those of common scab of potato and clubroot of crucifers.

The potato-scab organism is not so abundant nor is the disease so severe in soils with a pH of 5.2 or below. Severity of infection increases up to about pH 7.5, above which there is a tendency for infection to be reduced provided the pH of the soil solution remains constantly at this level. In practice it has been found that scab is not a serious problem in soils which have a natural reaction around pH 5.0. Fortunately potatoes grow well at this reaction. Avoidance of scab in highly acid soils can be assured indefinitely if soil and crop management is such as to keep the pH below 5.2 (Fig. 176).

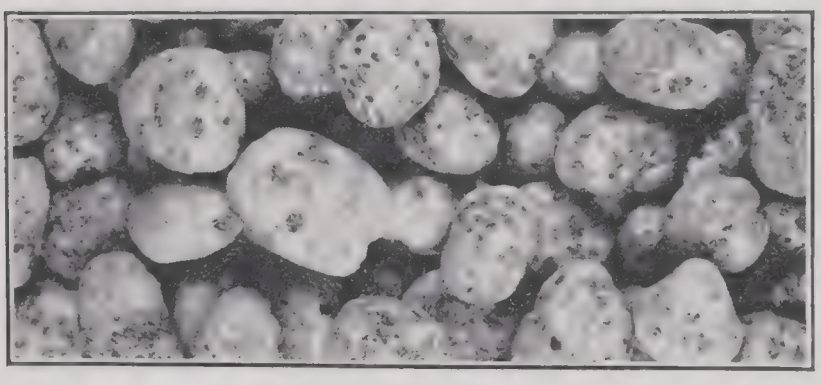
The crucifer-clubroot organism is inhibited by media which have a reaction of about pH 7.0 or above. Spores germinate well in acid solutions but poorly or not at all in alkaline solutions. In soils which have a reaction of the soil solution at pH 7.0 or above, the fungus does not ordinarily cause infection, but certain exceptions exist which will be discussed presently. In naturally alkaline soils clubroot seldom occurs. Since much of the culture of cabbage, cauliflower, and other crucifers is on soil below pH 7.0 in reaction, clubroot is a common disease. For centuries the application of lime to reduce the disease has been practiced. The success of this procedure is variable, depending on the buffer action of the soils. In some muck soils with high buffer action very heavy applications of lime have little effect. In some loam soils lime applications heavy enough to cause a soil reaction of pH 7.0 or above do not always prevent clubroot. When heavily limed soil on which the disease was not controlled in the field was brought into the greenhouse, where cabbage plants were grown upon it under the usual program of daily watering, infection was completely inhibited. If the soil moisture was kept at a fairly low level but allowed to fluctuate somewhat, infection did occur. In moist soil aerated by a continuous flow of air introduced into it through a perforated pipe, infection also occurred. These facts suggested that possibly the soil solution immediately adjacent to the roots (rhizosphere) had a lower pH than the soil mass, owing to the carbon dioxide given off by the roots. Thus germination of spores in this sphere might not be inhibited so that infection occurred, and once infection took place, the pH of the soil probably did not influence disease development. This set of conditions might be expected to occur more often in periods of alternating high and low soil moisture in the field, while in continuously moist soil under greenhouse culture the pH of the soil solution of the rhizosphere would probably be nearly the same as that of the soil mass.



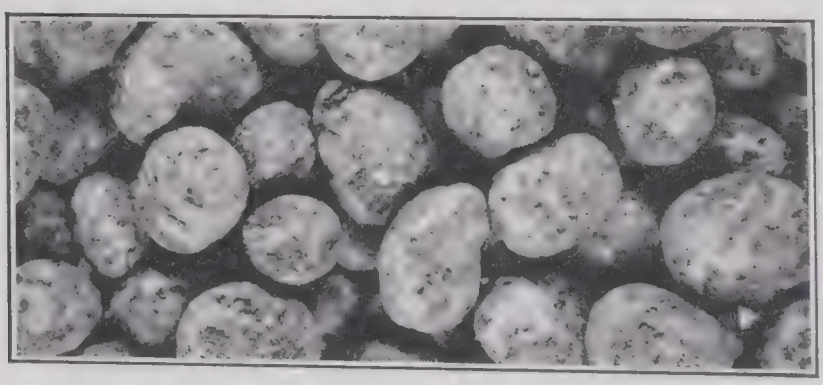
pH 4.4



pH 5.2



pH 6.2



pH 7.8

FIG. 176. The relation of soil reaction to the development of potato scab. Potato tubers grown on heavily infested soil in adjoining field plots in which the reaction of the soil had been adjusted to the pH levels indicated. Some disease occurred in each plot. It was least at pH 4.4, slightly more at pH 5.2, and continued to increase with decrease in acidity up to pH 7.8. (*Courtesy of R. H. Larson and A. R. Albert.*)

Soil Type. A few instances may be cited in which development of a soil-borne pathogen is influenced by soil type. It has already been mentioned that *Fusarium* wilt of cotton is common throughout the southeastern states as far west as eastern Texas. As cotton growing extended into the black limestone soils of Texas, wilt seldom developed in that region. On the contrary, *Phymatotrichum* root rot developed extensively in this area, and the pathogen seems to be favored by those soils. In Wisconsin

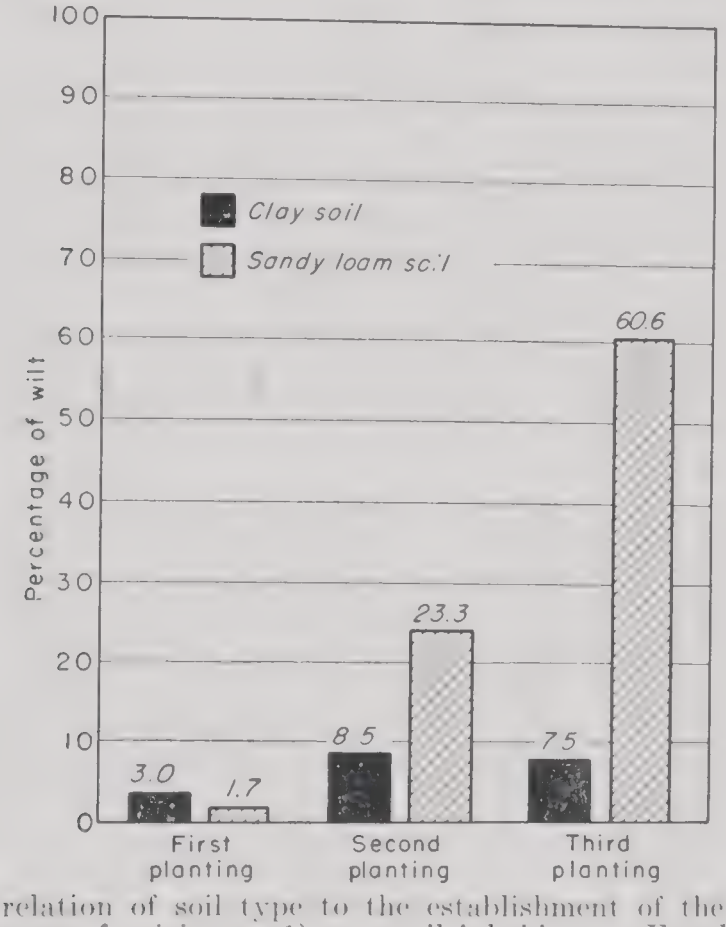


FIG. 177. The relation of soil type to the establishment of the pea-wilt fungus (*Fusarium oxysporum* f. *pisi*, race 1) as a soil inhabitant. Equal amounts of soil from an infested field were mixed with heavy clay and with sandy loam soil. Peas were grown in successive crops on the two lots of soil in the greenhouse under favorable conditions for the disease. Note that disease incidence increased rapidly in sandy loam soil and very little in the clay soil. (*After Walker and Snyder.*)

the pathogen of wilt of pea, *Fusarium oxysporum* f. *pisi*, race 1, develops most extensively in Miami and Carrington silt loams and less readily in Colby silt loam and Superior red clay and Superior clay loam (36) (Fig. 177). The root-rot organism, *Aphanomyces euteiches*, however, is favored by the last two types of soil, probably because of their higher water-holding capacity (p. 183).

Soil Fertility. There are many plant diseases in which the nutrition of the host has an effect upon the rate and degree of disease development. Many field experiments and controlled nutritional studies have been car-

ried out to determine the effects of various levels of nitrogen, phosphorus, or potassium and of the concentration of balanced nutrients upon disease. There are no definite rules which can be laid down. High nitrogen may increase severity of one disease and decrease that of another. Some diseases are most active on succulent rapidly growing host plants; others are most damaging to stunted undernourished plants. It is not often that a given disease may be controlled by application of a given type of fertilizer to the growing host crop, but the severity of some diseases may be lessened by certain types of fertilization.

Cotton wilt is commonly found to be more destructive to the crop when grown on potassium-deficient soils, and the application of high-potash fertilizer has real value in the reduction of wilt, particularly when used on resistant varieties. Tomato wilt and cabbage yellows have been shown in controlled nutrition experiments to be more severe in potassium-deficient than in balanced nutrient. Cabbage clubroot, on the other hand, is significantly less severe in potassium-deficient than in balanced nutrient. Increase in concentration of balanced nutrient reduces severity of tomato wilt and cabbage yellows but increases severity of cabbage clubroot and bacterial canker of tomato (Fig. 178). With pea wilt no effect of increase in nutrient concentration resulted in short midwinter days with low light intensity, but in longer days with higher light intensity wilt decreased with increase in nutrient concentration (35, 39–42).

Reduction of disease by change in balance or concentration of the nutrient in controlled greenhouse experiments does not always ensure control in the field by application of corresponding fertilizers. The interaction of other environal factors may reduce or entirely obscure the effect of fertilizer. Smith and Walker (31) found that they could completely control *Aphanomyces* root rot of pea in the greenhouse by increasing concentration of the nutrient. While heavy fertilization of naturally infested field soil reduced the amount of root rot during the first part of the crop season, the disease nevertheless caused severe loss when temperature and moisture were favorable (38). Thus caution should be taken in drawing up recommendations on the basis of one season's results, since another environment, another soil type, or a heavier infestation by the pathogen may result in a less encouraging outcome.

Predisposing Factors. Relatively little information is available regarding the influence of various soil and air factors in the environment upon the predisposition of the host to infection and disease development. In the main, as already indicated, the effects of environal factors have been recorded at the time of and following infection. The conditions of the soil for one or more crop seasons previous to the situation at hand are undoubtedly often important in the determination of the amount of inoculum avail-

Wilt

Canker

FIG. 178. The relation of mineral nutrients to the development of *Fusarium* wilt and bacterial canker of tomato. Plants at the left were grown in sand watered with a weak concentration of a balanced nutrient; those at the right were grown in a nutrient in which the concentration of salts was thirty times greater. In the upper pots plants were inoculated with the wilt organism; those in the lower pots with the canker organism. Note that wilt development is enhanced in the weak nutrient, while canker is greatest in the strong nutrient. (*Photographs by R. E. Foster and J. B. Kendrick, Jr.*)

able. Crop rotations usually remove the inoculum in the case of many plant pathogens in the class known as **soil invaders**, which do not become permanent members of the soil microflora and which often persist only as long as the host debris is available. With others which have very long-lived spores or which are in the class known as **soil inhabitants**, members of which become a more or less permanent part of soil flora, long rotations are essential and often inadequate. The type of crop residue, the amount of organic matter, the succession of previous crops, and soil management

FIG. 179. The effect of temperature on the predisposition of tomato seedlings to *Fusarium* wilt. Seedlings were grown for 50 days in pots in greenhouses maintained almost constantly at the temperatures indicated. They were then inoculated with the wilt fungus and all kept subsequently at 28°, the optimum for disease development. Note that plants grown at the lowest temperature were least favorably disposed to the disease and that plants were more favorably disposed to wilt as the preinoculation temperature increased up to 28°. (*Photograph by R. E. Foster.*)

as it affects moisture and reaction are all factors which may enter into the determination of the abundance of inoculum. In the main, however, we know relatively little about this subject.

The direct effect of soil factors upon the predisposition of tomato to *Fusarium* wilt was studied by Foster and Walker (11). This was done by growing tomato plants under a variety of conditions and then inoculating the roots and growing the inoculated plants under conditions very favorable for disease development. Tomatoes were found to be predisposed to wilt by (*a*) soil and air temperatures near the optimum for host-plant growth (Fig. 179); (*b*) low soil moisture; (*c*) short day length; (*d*) low light intensity (Fig. 180); (*e*) nutrient low in nitrogen, low in phosphorus, high in potassium, or low in pH. Susceptibility of the plants was decreased

and resistance was correspondingly increased by (*a*) soil or air temperatures above or below the optimum for plant growth; (*b*) very wet soil; (*c*) long daylight periods; (*d*) high light intensity; (*e*) nutrient high in phosphorus, low in potassium, high in nitrogen, and high in pH. Thus it is evident that

FIG. 180. Effect of light intensity on the predisposition of tomato seedlings to *Fusarium* wilt. The plants in the upper row were grown for 30 days at an average light intensity of 800 ft.-candles (*left*) and 90 ft.-candles (*right*). They were then inoculated with the wilt organism. In the lower row the same plants are shown 2 weeks after inoculation. (*After Foster and Walker.*)

the degree to which tomato plants are affected by wilt after transplantation into infested soil may be determined by the conditions under which transplants were grown as well as by the conditions which prevail after infection. Kendrick and Walker (16) studied bacterial canker of tomato in the same

way. Temperature and low soil moisture had predispositional effects opposite to those in *Fusarium* wilt, while the effects of high and low light intensity were the same, respectively, for each disease.

DISEASES INCITED BY AIR-BORNE PATHOGENS

It is obvious that the environment becomes more complex when the pathogen is one which passes through its life cycle above ground and in relation to the aerial portions of the host plants. The interaction of temperature and humidity is often most important. The development of inoculum and the local or widespread dissemination of the pathogen are important steps of the disease cycle which are commonly dependent upon environal factors. The relation of environment to the development and duration of periods of host susceptibility is sometimes critical. The relative importance of various factors differs with different diseases. The subject may be approached advantageously by a study of a few diseases in which epidemiology in relation to environment has been studied critically.

Leaf Rust of Wheat. This disease is important in the southern portion of the central plains area including Oklahoma and Kansas. The fungus, *Puccinia rubigo-vera tritici* (Eriks.) Carleton, overwinters as mycelium in fall-sown winter wheat with restricted increase of the uredial stage during the winter. The alternate host plays no part in overwintering of the fungus. The severe damage to the winter wheat crop is in the period from April until harvest in June. Chester (2) made a critical study of weather throughout a 23-year period in relation to rust development. He suggested in 1942 that leaf-rust epidemics might be predicted in Oklahoma by the end of March, about 10 weeks before harvest, at a time when leaf rust is ordinarily inconspicuous, but in time for growers to readjust plans if an epidemic was imminent. The minimum temperature for satisfactory urediospore germination is 10°C., and at this low temperature the incubation period is 3 to 5 days longer than at the optimum temperature. The temperature and precipitation during late winter are usually unfavorable to the rust in that area. It is necessary, however, for several generations of rust to occur in late winter (March) in order for enough inoculum to develop for an epidemic. Generally after early April moisture and temperature are within the optimum range for multiplication and spread. The critical period, therefore, is that just before April when inoculum is built up or retarded. In a "normal year" in Oklahoma there occurs about 1 rust pustule per 10 leaves on Mar. 1 with an increase to 1 pustule per leaf on Apr. 1 and about 1,000 pustules per leaf at maturity with a crop loss of about 5 per cent. In a mild moist winter the organism goes through successive cycles of urediospore formation unimpeded. In the epidemic

year of 1938, rust was very conspicuous on Apr. 2. The 1,000-pustule-per-leaf stage was reached at about heading time, and the crop loss was estimated at 29 per cent. Eight successive forecasts (1941–1948) were shown to be highly reliable (3).

Late Blight of Potato. This disease has been the subject of many studies of epidemiology in various parts of the world. For 100 years it has continued to harass the production of potato in spite of very satisfactory control procedures. In practically all cases the source of primary inoculum is the overwintering mycelium in seed tubers. The fungus invades the sprouts either after planting or in refuse piles from storehouses and under favorable conditions produces sporangia, which are readily carried long distances by air currents but are relatively short-lived. The sporangia germinate directly by germ tubes or indirectly by formation of about eight motile zoospores, which in turn become sessile and germinate by means of germ tubes. Infection proceeds by penetration of leaf or stem tissue, and the sporangial cycle is repeated under favorable conditions in a relatively short period. The presence of overwintering inoculum is usually assured. The development of an epidemic is dependent upon environment.

Crosier (5) has reported the most extensive analysis of the reaction of the pathogen to temperature and moisture of the atmosphere. The sporangia are formed on host lesions at a minimum relative humidity of 91 per cent (optimum of 100 per cent) and at a temperature range of 3 to 26°C. (optimum 18 to 22°). The sporangia formed at 15° reach an indirect germination peak in 1 to 2 hr. at favorable temperature, while those produced at 25° reach the indirect germination at 5 to 7 hr. The optimum temperature for the indirect germination of the conidia is 12°; for direct germination, it is 25°. The zoospores germinate best at 12 to 15°. The germ tube grows best at 21 to 24°. It is obvious that the best conditions for the rapid completion of the life cycle of the fungus consist in plenty of moisture, cool nights, muggy days of moderate temperatures, all with a continuity sufficient to allow the parasite to keep multiplying. With such factors in the prevailing weather an epidemic develops. The weather requirements explain the annual occurrence and almost endemic nature of the disease in the countries surrounding the North Sea and in the upper New England states. The disease is restricted in northern Norway because average temperatures are too low.

In view of the wide occurrence of the potato crop and the importance of late blight, forecasting services have been worked out in some countries, *e.g.*, Holland. By watching the weather critically it is possible to predict late-blight epidemics in time to warn growers regarding the necessity for more frequent application of sprays than is usual. In Holland the weather

requirements for a late-blight epidemic as used in the forecasting service are as follows:

1. Night temperature below the dew point for 4 hr. or more.
2. Night temperature not below 10°C.
3. Mean cloudiness not below 0.8 on the following day.
4. Rainfall of at least 0.1 mm. on following day.

In the United States a forecasting service for late blight was instituted in the upper Mississippi valley in 1943 (26). Here average temperatures below 21°C. (70°F.) during June and July were taken as the most important criterion for development of inoculum sufficient to bring on an epidemic with continuance of favorable temperature and humidity in August and September. In eastern Virginia a forecast of late blight was made when rainfall exceeded a critical line and the average daily temperature remained below 24° for two successive weeks (4). It is agreed that much more detailed evaluation of other factors than rainfall and temperature must be worked out before a reliable basis of forecasting late blight is attained, and such a basis will probably differ with the growing conditions of localities concerned (44).

Downy Mildew of Cabbage. The causal organism of this disease, *Peronospora parasitica* (Fr.) Tul., has many physiologic races. The race which is pathogenic on cabbage and other members of the cole group is widespread in the United States and is ordinarily of minor importance, appearing as a pathogen of the lower older leaves of the plant. When conditions are favorable for its development on young seedlings, it may be very damaging, since the hypocotyls are invaded rapidly and the plants are predisposed to facultative parasites. Felton and Walker (8) have shown that conidia germinate most rapidly at 8 to 12°C. Penetration of the host occurs most rapidly at 16° (Fig. 181). After infection mycelium grows most rapidly at 24°. Lesions develop most rapidly at 24°, but sporulation and reinfection are sparse at this and at higher temperatures (Fig. 182). At 16° the growth of the host and the fungus is slower, but sporulation and reinfection are much more pronounced than at higher temperatures. Here the fungus is most prolific, and the disease develops most profusely. Apparently the low optima for sporulation, germination, and penetration are more important to disease development than the higher optimum for growth of the pathogen. The disease is found to be most destructive in seedbeds in southern states during winter months when the temperature averages from 10 to 15°. This seems to be explained best by the effect of temperature upon production of inoculum, spore germination, and infection.

Apple Scab. This disease is world-wide in its occurrence and one of the most important on apple in many regions. It has been studied critically

in Wisconsin by Keitt and Jones (14). The most important means of overwintering of the fungus, *Venturia inaequalis*, (Cke.) Wint., is as various stages of immature perithecia in dead leaves on the ground. Primary infection in the spring occurs from ascospores produced in perithecia,

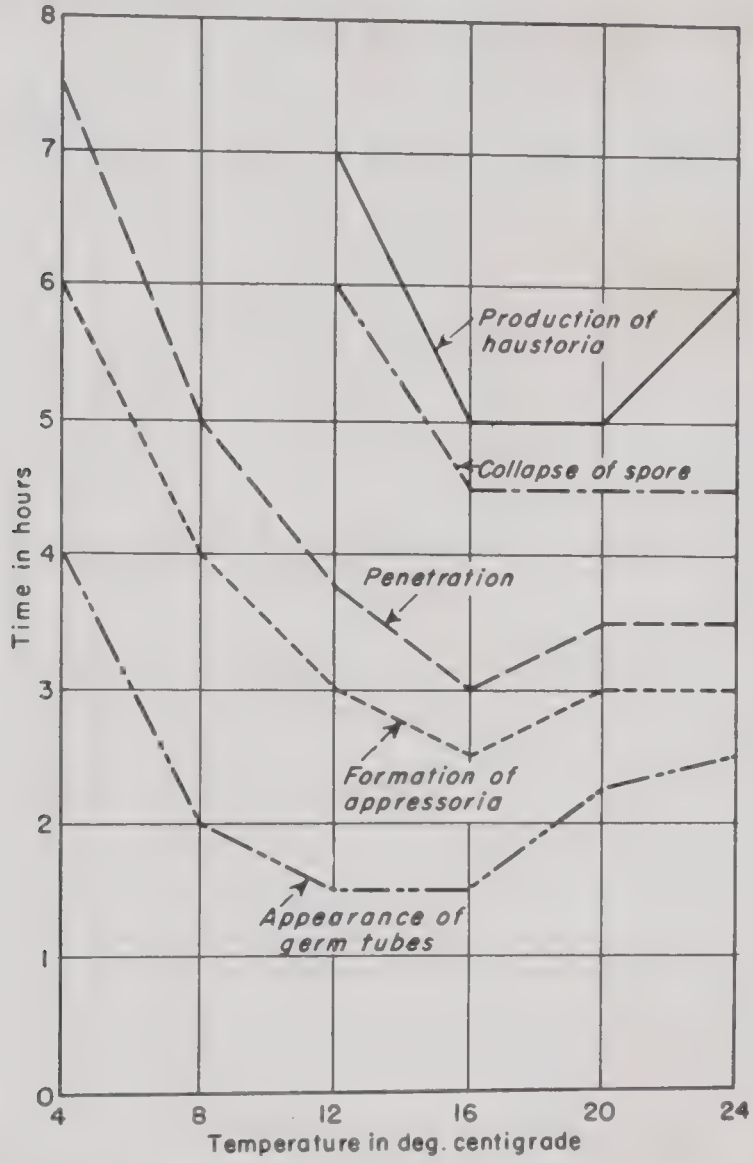


FIG. 181. The relation of temperature to the germination, formation of appressoria, penetration, and production of haustoria by the cabbage downy mildew fungus. Note that the first three processes proceed most rapidly at 16° or below, while haustoria form first at 16° and 20° (compare with Fig. 182). (*After Felton and Walker.*)

which are discharged into the atmosphere and carried to apple foliage by air currents. Early growth of perithecia is favored by relatively low temperatures (optimum about 13°C.). Maturation of ascospores is optimal at about 20°. At 24° ascospore development is retarded in moist leaves. Ascospores are discharged chiefly during rains over a period of several weeks. The amount of primary inoculum is, therefore, influenced greatly

by duration and frequency of spring rains. Leaves and other green parts of the host are most susceptible when young, and they pass relatively rapidly with growth into a stage resistant to the scab fungus. The fruit buds are the first to open, those on fruit spurs generally in advance of those on terminal shoots. Apical portions of sepals and leaves are the first susceptible parts exposed to ascospores. If weather is rainy at this

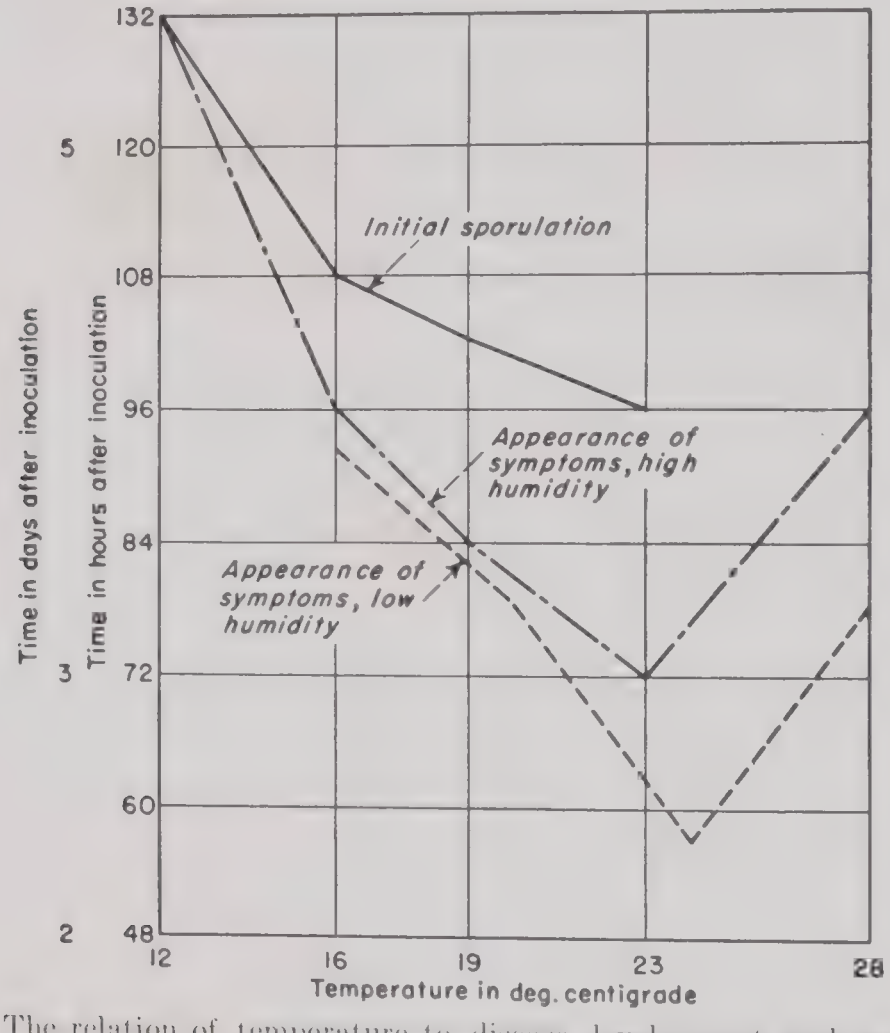


FIG. 182. The relation of temperature to disease development and sporulation of the cabbage-downy-mildew fungus. Note that these processes occur most rapidly at 23° (compare with Fig. 181). (*After Felton and Walker.*)

time, infection of sepals is abundant, and this is particularly favorable to early and continued secondary infection. On the other hand if weather is unusually warm and dry, ascospore discharge is prevented. Moreover, the host growth being more rapid, sepals and leaves grow into the resistant phase with a minimal amount of infection. Secondary infection of young leaves and fruits is by means of conidia which are produced freely on very young lesions. Conidia are not detached readily except by water, and they are therefore dependent upon rain for dissemination. The most critical period for the development of epidemics is from the time the sepals are

first exposed until some 2 to 4 weeks after petal fall. If this period is cool and rainy, prevention of the disease by fungicides is the more difficult because conditions are most favorable for ascospore discharge and the exposed sepals and leaves pass slowly through their susceptible period.

Cherry Leaf Spot. The epidemiology of this disease has been studied by Keitt *et al.* (15) in the sour-cherry-growing region of northeastern Wisconsin. The fungus (*Coccomyces hiemalis* Higgins) overwinters in infected leaves of the previous season as stromata in which mature apothecia bearing ascospores are produced in the spring. Spring rains are essential for ascospore production and discharge. The air-borne ascospores serve as the primary inoculum. In cherry the period of susceptibility is the reverse of that of apple in relation to the scab fungus. Young leaves are not infected; they become more susceptible as they grow toward maturity. At the time of maximum ascospore discharge, therefore, there is relatively little vulnerable host tissue. The first tissues available are the inner scale leaves of buds and the calyxes of blossoms, but both are shed rather early. Primary infection is therefore ordinarily sparse and serves chiefly to establish the fungus. Conidia are produced in acervuli and develop readily under moist conditions, except at high temperatures, and, since they form in a gelatinous matrix, are resistant to adverse conditions of heat and drought for long periods. They are disseminated chiefly by water. Thus periods of rain are essential to secondary infection, and the epidemic development of the disease is influenced most by the relative abundance of rain during the summer period. The sooner the disease develops in this period, the greater the damage to the host. The fungus requires comparatively long moist periods to bring about abundant infection.

Potato Mosaics. The virus disease of potato known as mild, or crinkle, mosaic is a serious disease on certain varieties of potato, one of the most important in the United States being Triumph. It was commonly observed that seed from fields in northern states which appeared to be quite healthy yielded severely diseased plants when grown in southern states in cool winter months. This was shown to be due to the fact that the symptoms were masked during the warmer weather of the crop season in the North. Tompkins (34) studied the effects of air temperature, soil temperature, soil moisture, humidity, light, and nutrition on the development of this disease. He found air temperature to be the only factor which affected the expression of mosaic symptoms in diseased plants. Exposure of diseased plants to air temperature of 23 to 24°C. and above resulted in masking of disease symptoms. Relatively short exposures daily to these high temperatures were sufficient to cause masking. Thus in northern summers when the air temperature rose to 23° for 1 or 2 hr. daily, the failure of the disease to appear was explained. A second mosaic disease, latent mosaic,

in which symptoms occur only on relatively new varieties in the country, is also favored by cool temperatures. A third mosaic disease of potato, rugose mosaic, is, on the contrary, retarded by cool temperatures and enhanced by warm temperatures (p. 506).

Cabbage Mosaic. This disease is ordinarily the result of infection of the host by two viruses, one of which is a strain of turnip virus 1 and the other a strain of cauliflower virus 1. The first of these is responsible for mottle symptoms, the second for veinclearing symptoms. Various other symptoms occur as the combined effects of the two viruses. In nature the mottle symptoms predominate during warm weather, and veinclearing appears more prominently during cool weather. A critical study of the disease has brought out the interesting fact that the two viruses respond differently to temperature. The concentration of the mottle virus is greatest in the tissues of cabbage and the pathological effects most pronounced at 24° and 28°. At 16° the mottle virus is less abundant, and mottle symptoms are usually masked, while at this temperature the veinclearing virus is the more abundant (28).

RESTRICTION OF DISEASES BY CLIMATE

In the diseases discussed up to this point in this chapter the relation of environment to infection and disease development has been considered. We may now consider some cases, including a number already discussed in previous chapters, in which regional distribution is restricted by the inimical effects of one or another factor in the environment. These factors vary. They may preclude the overwintering of the pathogen; they may restrict the build-up of inoculum; or they may effectively prevent infection.

It has been shown that apple scab is rare in California. This may be due primarily to lack during the growing period of sufficient rainfall to disseminate conidia. Bean anthracnose is practically nonexistent west of the Continental Divide. The pathogen requires spattering rain for dissemination of spores, and since the crop is practically always growing in low-rainfall periods, establishment of the disease is prevented. Practical application of this fact was made when most of the bean-seed production in the United States moved into this area, assuring anthracnose-free seed.

In the Pacific coast areas generally, black rot and blackleg of cabbage are rare. The chief reason for this is that the plants are grown in seedbeds during the low-rainfall season. Dissemination and establishment of both pathogens depends upon rainfall. Thus although the plants, when grown for seed, pass later into a favorable climatic period for the pathogens, the fact that the latter fail to become established earlier precludes their development during favorable environment. Another cabbage disease, ring spot, *Mycosphaerella brassicicola* (Fr.) Lindau, thrives in Pacific coast regions

(27). The pathogen is an Ascomycete which, having no conidial stage, depends entirely upon air-borne ascospores for dissemination and infection. Epidemics develop during the rainy season, and seed becomes infected. This fungus has not been found on growing plants in the United States east of the Cascade and Sierra Nevada mountains, although thousands of pounds of seed, much of it infected by the pathogen, is sown annually in eastern United States. Although the limiting factor or factors which prevent establishment of the fungus in the latter region are not fully understood, it is undoubtedly due to the environment. In other words the environment west of the Cascades and Sierra Nevadas favors ring spot and prevents blackleg and black rot, while the climate east of the 100th meridian often favors blackleg and black rot but consistently prevents ring spot.

The restriction of pathogens by the fact that they are unable to survive northern winters is not uncommon. The root-rot disease, *Phymatotrichum omnivorum* (Shear) Duggar, of cotton and many other plants is restricted to a region bounded on the north roughly by the latitude of southern Oklahoma. Ezekiel (6) has shown that the fungus in any form survives freezing temperatures for only a day or two. The northern border of natural occurrence coincides relatively closely with that at which the lowest observed temperature is −10°F. (−23°C.). It is quite probable that the fungus in its present form will not move north of the present root-rot area. Other very important pathogens restricted to southern states are *Pellicularia rolfsii* (Curzi) West and *Pseudomonas solanacearum* (E. F. Sm.).

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CHAPTER 14

HOST–PARASITE INTERACTIONS

The physiology of parasitism is concerned with the interaction of two biological entities. This interaction cannot be separated from the interplay of enviromal factors upon it. In the preceding chapter the various effects of environment before and after infection upon host and parasite and upon disease development were considered. In this chapter we shall be concerned with the morphological and physiological aspects of the intimate contact of host and pathogen during the various steps in pathogenesis. The processes concern the most fundamental problems of plant pathology, and their elucidation must go hand in hand with our understanding of cell metabolism.

The subject is taken up in three major divisions. First the relation of the pathogen to the host tissue is presented to compare the various ways in which pathogenesis is brought about. Second, the various defensive mechanisms which host varieties and individuals contain which contribute to resistance are discussed. Third, the variability of pathogenicity in microorganisms and some of the mechanisms underlying such variability are considered.

RELATION OF PATHOGENS TO HOST TISSUE

Most bacterial and fungus pathogens have rather specific means by which they penetrate the host and relate themselves to the host tissue. In this section some of the common methods of invasion and disease development will be discussed. Many examples will be found among the specific diseases described in preceding chapters.

Penetration

Through Natural Openings. *Stomata* on aerial portions of the green plant serve as channels of entry of many fungi and bacteria, *e.g.*, the rust fungi, bacterial blights of bean, bacterial canker of tomato, fire blight of pear and apple. While the mycelium of fungi may grow through the stomatal opening or in some cases force a way through closed stomata, bacteria are somewhat more exacting in their requirements. Films of moisture extending from the exterior through the stomatal aperture to the substomatal chamber are apparently essential. It is assumed, without any direct proof, that bacteria enter in the motile form. However, it is well to point out that under favorable conditions of high humidity, required as well by motile forms, nonflagellate species, such as the tomato-bacterial-canker organism, invade stomata about as readily as do organisms with

flagella. The advantage of water in the substomatal chamber and in the aperture is shown by the work of Johnson (54). When the substomatal chamber is filled with water by artificial means, both fungi and bacteria invade more readily, while some organisms cause incipient infection on hosts which they do not infect in nature. *Hydathodes* are not as common channels of entry as are stomata. In some cases, however, they are important. The bacterial pathogen of black rot of crucifers enters the host largely through hydathodes, although it may invade through stomata of cotyledons and through insect wounds. *Lenticels* are points of invasion of some fungi and some bacteria. Under special circumstances the soft-rot bacteria may enter lenticels of potato tubers, while the potato-scab organism enters commonly in that way. The brown-rot fungus invades through the lenticels of immature plum fruits. *Root ruptures*, particularly the rupture of the cortex by secondary root primordia, provide a channel of entry to some root pathogens. The tobacco-black-root-rot fungus, *Thielaviopsis basicola* (Berk.) Ferr., usually invades through such openings (23). *Gibberella zeae* commonly enters the corn plant through root ruptures. *Rifts in the cuticle* may occur in some cases. Penetration of the cuticular layer of bean cotyledons by bacterial-blight pathogens follows swelling of the seed during germination. The bacteria are shown to move en masse through the natural breaks which occur during imbibition of water (Fig. 183).

Through Wounds. Many types of wounds occur on surfaces of plant parts. Some organisms which penetrate directly or through natural openings may enter wounds when they are available, but such is not always the case. Many organisms depend entirely on wounds for penetration. The soft-rot bacteria usually invade through wounds. Some facultatively parasitic fungi invade only under similar circumstances. *Abrasions, bruises, freezing injury, sunscald injury* are common types of wounds which predispose the plant to these organisms. *Insect wounds* are essential to some bacteria and fungi (59). The cucurbit-wilt bacteria invade entirely through wounds created by cucumber beetles. The Dutch-elm fungus depends on the wounds of bark beetles for infection. Flea beetle injury may increase the incidence of early blight on potato and tomato, although the causal fungus normally penetrates directly. *Wounds produced by wind-blown dust* are the most common points of entry of the immature tomato fruit (where stomata are not present) by the bacterial-spot pathogen, *Xanthomonas vesicatorium* (Doidge) Dowson (34). The tomato-anthracnose fungus, *Colletotrichum phomoides* (Sacc.) Chester, may take advantage of this type of wound (57), although it may also penetrate the cuticle directly (33). Wounds afforded by breaking of epidermal hairs may also serve as a channel of entry as in the case of bacterial speck (16).

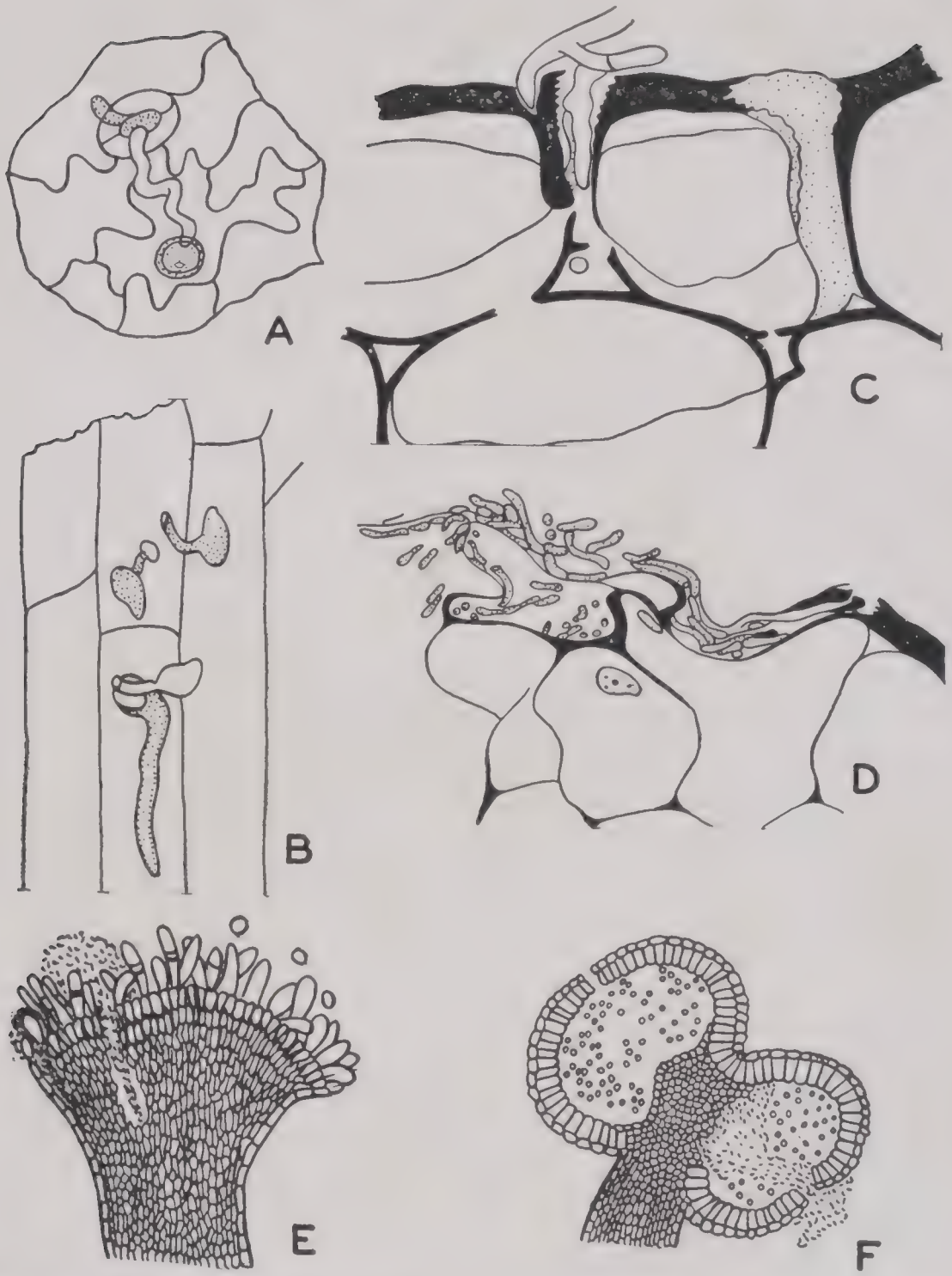

FIG. 183. Penetration of the host. *A*, stomatal penetration by the dicaryotic mycelium arising from urediospores of the bean-rust fungus (*Uromyces phaseoli typica* Arth.) through the stomata of the bean leaf; *B*, the monocaryotic mycelium arising from the sporidia of the same fungus penetrates the same host directly; *C*, mycelium of *Gibberella zeae* penetrating the epidermis of the unruptured coleorhiza through a plug between two epidermal cells; *D*, penetration by the same fungus through the margin of a rupture in the mesocotyl produced by root emergence; *E*, Penetration of fire blight bacteria through the stigma of the pear flower; *F*, penetration of the same organism through the anther of the pear flower. (*A*, *B after De Bary; C*, *D after Pearson; E*, *F after Hildebrand.*)

Direct Penetration. Kühn and De Bary were the first to observe that pathogenic fungi sometimes penetrate the host directly through the outer host wall. Kühn (58) showed this to be the case with wheat bunt; De Bary (26) described it with white rust; Ward (91) figured direct penetration of the potato leaf by the late-blight organism. De Bary (27) was the first to study critically the nature of direct penetration by *Sclerotinia sclerotiorum*. He laid emphasis on the relation of nutrient in the infection drop (*i.e.*, the liquid on the outer host surface which contained the organism previous to penetration). If the infection drop contained suitable nutrient, the fungus penetrated readily. If the drop consisted of water with little or no nutrient, the fungus first produced an appressorium, which in turn produced toxic material. The latter diffused into the host tissue, killing some of the cells, from which nutrients in turn diffused back into the infection drop. The nutrients converted the fungus from a saprophytic to a parasitic phase. This interpretation was challenged and disproved many years later by Brown (8–12). He showed that electrolytes did diffuse from the host cells to drops of water on the surface. The materials might stimulate or retard growth of fungi in the drop, but there was no evidence of diffusion of toxic products of the fungus through the cuticle until it had been penetrated by the fungus. No conclusive evidence of production of cutin-dissolving enzymes by fungi or bacteria has been produced up to the present. Blackman and Welsford (5) were the first to clarify the mechanics of penetration of the cuticle. They demonstrated that the outer wall of the appressorium (and sometimes that of the hyphal tip) was surrounded by a gelatinous layer which served to cement the organ to the host surface. A small hypha growing from the side of the appressorium in contact with the host penetrated the cuticle, and once beneath the latter, the hypha grew to normal width (Fig. 184).

Miyoshi (66), working in Pfeffer's laboratory, had previously studied the physiology of penetration. He studied growth of germ tubes in drops of water in which had been placed capillary tubes containing various concentrations of nutrients. The effect of diffusion of materials through membranes or punctured mica was also studied. From these investigations came Miyoshi's chemotropic theory, which postulated that penetration was determined by whether or not the materials in the host cells beneath the membrane were positively chemotropic to the fungus. Fulton (32) failed to confirm Miyoshi, while Graves (38) lent some support to the conclusion of both Miyoshi and Fulton. Brown and Harvey (15) reopened the study and dispelled rather completely the place of chemotropism in the infection phenomenon. They found that *Botrytis cinerea* would penetrate an onion skin membrane whether or not all nutrients were previously removed by prolonged washings. Miyoshi (67) had early described puncture of gold foil and paraffin. Brown and Harvey also showed that penetration of

paraffin-wax membranes devoid of nutrient occurred readily. They concluded that the major stimulus to penetration was a contact stimulus rather than a chemical one.

It is well to point out here that some plant surfaces are not cuticularized

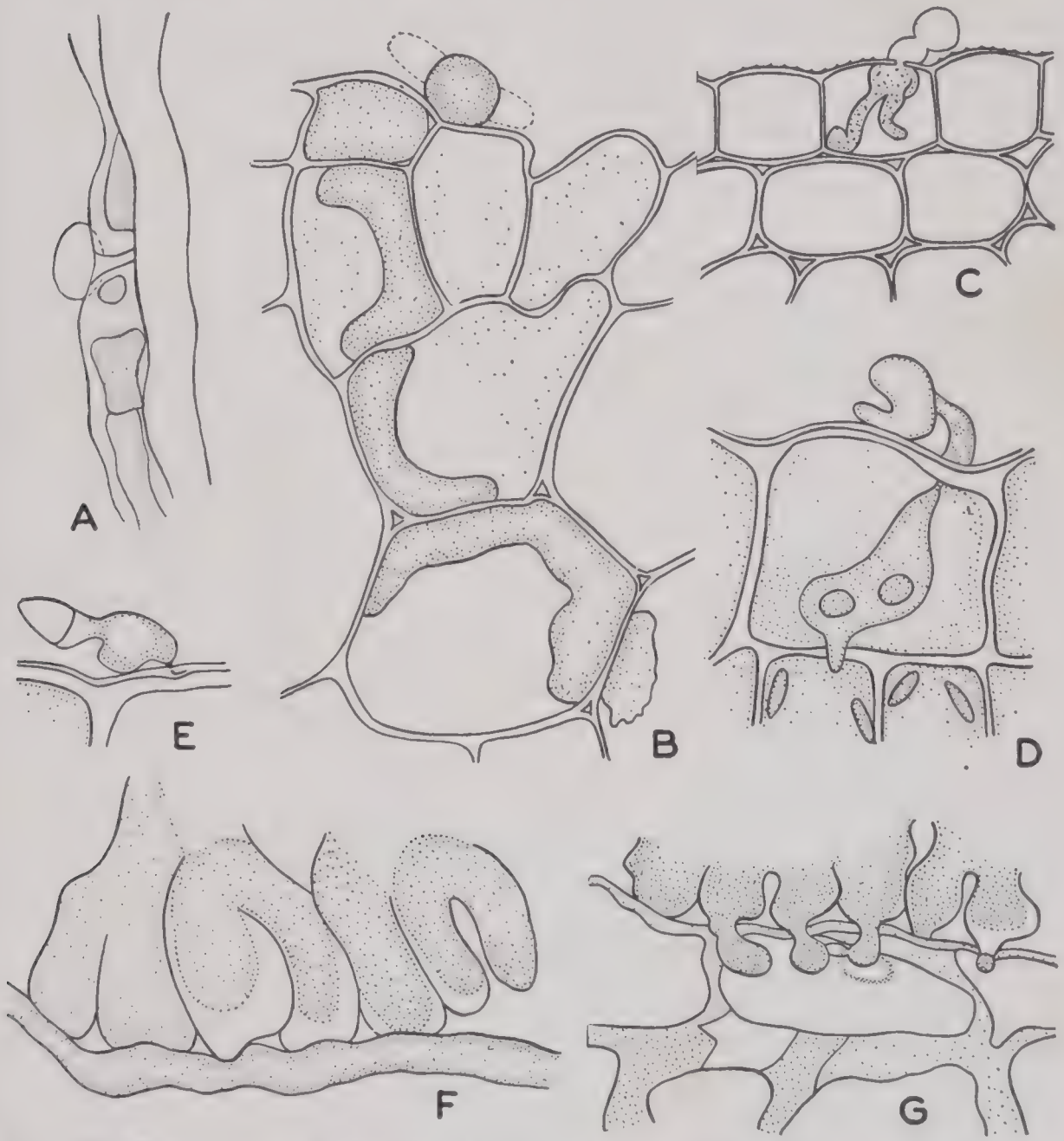

FIG. 184. Penetration of the host. *A*, penetration of onion scale by *Colletotrichum circinans*. The penetration tube arising from the appressorium pierces the cuticle of the epidermal wall and develops first between the cutin layer and the cellulose wall, later digesting the latter. *B*, penetration of bean leaf by *Colletotrichum lindemuthianum*. The conidium is shown in outline close to the appressorium. The penetration tube pierces the cuticle and dissolves its way promptly through the cellulose wall and develops into intracellular primary mycelium. *C*, penetration of potato leaf by *Phytophthora infestans*. *D*, penetration of young apple leaf by *Gymnosporangium juniperi-virginianae*. *E*, penetration of old apple leaf by the same fungus at a stage when the leaf has become resistant. *F*, *G*, development of appressoria and penetration of broad-bean leaf by the watery-soft-rot fungus (*Sclerotinia sclerotorium*). (*B after Leach; C after De Bary; D, E after Nusbaum; F, G after Boyle.*)

or suberized. This is true for some embryonic or immature organs, root hairs, some parts of fruits. Curtis (25) noted that some varieties of plums had patches of uncuticularized surface through which *Sclerotinia fructicola* invaded readily. Root hairs and root tips are common portals of entry by many organisms, *e.g.*, the vascular-wilt fusaria, the clubroot organism, and the nitrogen-fixing bacteria. Pearson (72) showed that *Gibberella zeae* penetrated the unruptured corn coleorhiza at points where plug-like deposits between the epidermal cells were dissolved by fungus enzymic action.

Development Following Penetration

It should be emphasized that, as already pointed out on p. 6, penetration does not necessarily imply infection. Johnson (53) showed that the onion-smudge organism penetrated directly many plant species which did not become hosts, because no further development occurred. Gibson (37) showed that the chrysanthemum-rust fungus penetrated the stomata of many plant species in which no infection followed. Bond (6) reported similar results with quite a distinct fungus, *Cladosporium fulvum* Cooke. Schroeder and Walker (78) found that certain vascular fusaria, although noninfectious to pea, penetrated pea roots and invaded stems up to the third or fourth node without causing any signs of disease. The establishment of infection requires more than penetration. It is the interaction of host and pathogen which determines the type of reaction which follows. This is usually a complex process, and while certain groups of pathogens are characterized by a certain type of reaction, there is no general pattern for pathogens as a whole. The basic nature of host-parasite interactions is still imperfectly understood. The metabolism of the living cell is still one of the great challenges in biology, and the intricacies of the interaction of two quite distinct cells or groups of cells are still more difficult to unravel. By cytological, histological, physiological, and biochemical approaches, parts of the picture in many situations have been delineated. Much remains to be determined, and progress in this problem is intimately associated with progress in our understanding of normal cell metabolism and growth.

Some of the cases already discussed in previous chapters and others which will now be cited will serve to illustrate the types which are now recognized (Fig. 185).

Digestion of Host-cell Walls. The works of De Bary (27) on *Sclerotinia* and of Ward (92) and Nordhausen (68) on *Botrytis* dealt with cases in which a major aspect of fungus action after penetration was the secretion of enzymes which diffused in advance of the pathogen and brought about a breakdown of portions of the cell membrane. Brown (8–10) added much

new information to the picture with *Botrytis*, as a result of further study with improved techniques. The major enzyme was originally called cytase, later pectase, and more recently propectinase. Its chief action was to dissolve the pectic materials of the middle lamella. The same enzyme was described in connection with the soft-rot bacteria by Jones (56) and by Van Hall (84). The common initial result is a macerating effect on the tissue, loosening the cells from one another and setting up various degrees of exosmosis. As already pointed out with bacterial soft rot, watery soft rot, and *Rhizopus* soft rot, the extensive withdrawal of liquid from the cells leads to a watery or mushy soft rot. In some cases, as in onion neck rot,

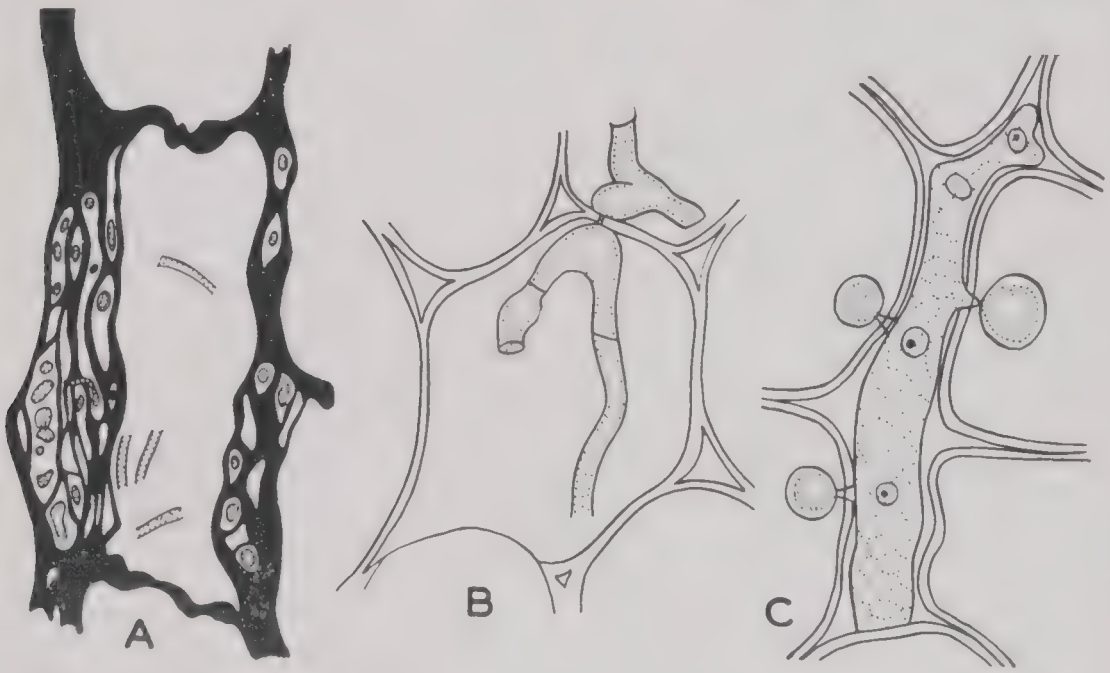

FIG. 185. Relation of fungi to the host tissue. *A*, digestion of the onion cell wall by *Colletotrichum circinans; B*, penetration of the corn cell wall by a greatly constricted hyphae of *Gibberella zeae; C*, intercellular mycelium and haustoria in cells of shepherd's-purse infected by *Albugo candida*. (*B after Pearson; C after Wager.*)

the same or a very similar enzyme is formed, but the mushy rot does not necessarily follow as completely. The yield of propectinase by a given organism is influenced by the substrate (31). It is important to remember that in this type of parasitism the real pathogenic action is in advance of the organism, while the latter follows in an almost purely saprophytic existence.

The next usual effect is that of cytolysis, *i.e.*, killing of the cells. In soft rot the first step is plasmolysis, which normally proceeds until the reaction is irreversible and the cell dies. De Bary (27) believed that the wall-dissolving and the lytic processes were distinct and were probably brought about by different enzymes. Brown (8–10), after a thorough study of the case with *Botrytis*, concluded that only one enzyme was concerned and that

the wall solution and lysis were stages of a progressive series of events. In this group of pathogens dissolution of the pure cellulose membranes was absent, or at least it was so minor as to elude identification. While thermostable toxic materials, *e.g.*, oxalic acid, have been regarded as the most important by some investigators (79), the evidence in that direction is not convincing. However, there are some clear-cut cases of toxic agents other than enzymes reported in other diseases. Among these may be cited the toxic agent associated with the tobacco-wildfire organism (21), oxalic acid formed by *Penicillium oxalicum* Currie & Thom in seedling blight of corn (52), and a thermostable toxic material produced in culture by *Pellicularia rolfsii* (Curzi) West (46). Brown (14) regards the last three cases as needing further study to determine whether the toxic materials are actually present in the region where the pathogen is invading. Further, it is well to refrain from assuming in cases cited earlier in this section that propectinase is the sole functional material in pathogenicity. The work of Harter and Weimer (43) is particularly apropos in this connection. They investigated a number of species of *Rhizopus* parasitic on sweet potato with reference to their production of propectinase in pure culture. The most active pathogen, *R. nigricans* Ehr., was one of those producing the lowest yield of propectinase, while the species producing the largest amount was a very weak pathogen. It is obvious that other unknown factors than propectinase enter into the pathogenetic complex in this group of organisms, which are generally regarded as having one of the simplest types of pathogenicity.

Wilt Production. While wilt, or loss of turgidity, is a common symptom of disease, it may result from one of many possible causes. The so-called vascular wilts are set off as a group because of the association of symptoms with the presence of the pathogen in the vascular system of the host, usually in the xylem. The most common pathogens are the vascular fusaria, *Verticillium* spp., and vascular bacteria, *e.g.*, the pathogens of cucurbit bacterial wilt and southern bacterial wilt. Many symptoms comprise the disease picture for any one of these, *e.g.*, veinclearing, epinasty, chlorosis, vascular discoloration, stunting, temporary or permanent loss of turgidity, and death. A common factor, also, is that the various symptoms appear at some distance in advance of the parasite.

Various theories have been advanced to explain wilting. Plugging of the xylem by the pathogen, though possibly a factor in some of the vascular bacteria, has not been a satisfactory explanation for most fungus wilts. Many investigators have shown that the filtrates from cultures of wilt fungi on liquid media contain substances which cause wilting of plants when cut stems are inserted in the liquid. While this common phenomenon suggests that the fungus secretes a toxic material when present in the host,

there are numerous objections to such an explanation. Essentially the same results can be secured with filtrates from cultures of pure saprophytes. Furthermore, the filtrates of pathogens are nonspecific in their effects on host plants, although most of the organisms are very specific in their pathogenicity. While wilting of plants placed in the filtrates occurs, the complete syndrome of disease symptoms produced in the living plant, when parasitized naturally, is usually absent. Thus it is not at all certain that the culture filtrates contain the actual substance or substances responsible for the disease in nature. On the other hand, the fact that the action is produced at a considerable distance in advance of the fungus indicates strongly that some metabolite is concerned.

With certain of the fusarial wilts, *e.g.*, pea wilt as studied by Linford (61), it is shown that diseased plants transpire more water than healthy plants, indicating that wilt, rather than being the result of plugging, is on the contrary due to excessive water loss. It is further suggested that one of the major effects of vascular invasion and distribution of metabolic products of the fungus by the xylem stream may be a change in permeability of the host cells generally, leading to more rapid water loss. Gaümann and Jaag (36) have presented evidence to support the conclusion that in fusarial wilt of tomato the metabolic product responsible for the permeability change is a polypeptide, lycomarasmin. Hodgson *et al.* (47) have shown that a considerable list of substances of high molecular weight will cause wilting when they are permitted to enter the vascular system in proper concentrations.

All vascular wilts do not follow the same transpirational pattern. A study of two bacterial wilts, cucurbit wilt and Stewart's wilt of corn (*Bacterium stewarti* E. F. Sm.), is of interest in this connection. Harris (40) found that beginning with the first sign of wilt in the case of each disease, the transpiration rate declined progressively as compared with uninoculated controls, indicating that the major effect of infection was mechanical obstruction of water flow in the vessels.

Necrosis. In many diseases already described, necrosis of leaf, stem, or root tissue is the prominent disease symptom. The leaf spots, foliage blights, and root rots are common examples. While we know very little about host-parasite relations in this group of diseases, some cases have been studied rather extensively by means of histological techniques. In bean anthracnose the relation of the pathogen to the susceptible host is of interest. The spore germinates, and as the germ tube contacts the host cuticle, an appressorium is formed. An infection peg penetrates the cuticle, and the hypha then dissolves the epidermal wall to invade the epidermal cell lumen by the shortest route. In the cell the mycelium flourishes and increases in diameter without killing the host cell (Fig. 184). As the fungus

contacts the cell wall opposite the outer wall, it develops an appressorium-like body and penetrates by a relatively small peg. By enzymic or mechanical action or both, it invades the next cell, where a similar host-parasite relation is resumed. This so-called "primary mycelium" continues until several contiguous cells are so occupied. Up to this point there is an approach to a congenial symbiosis between the protoplasts of host and parasite without any lytic effect on the former. Then, more or less abruptly, development of "secondary mycelium" follows, in which case hyphae of smaller diameter ramify rapidly through and between cells. Host cells then collapse promptly; the necrosis becomes macroscopic by darkening of the lesion; and the "spot" takes on a characteristic delimited configuration.

Just why, with a given fungus, the lesion develops in a characteristic manner for a characteristic spot in size, shape, and delimitation is not known. In some diseases the spots are small and circumscribed by definite margins; in some they are spreading and indefinite. While in many of the cases studied there is evidence of enzymic action on the host-cell wall, *e.g.*, in onion smudge (Fig. 185), most leaf-spot and blight diseases show relatively little such action in comparison with the soft rots and *Botrytis* diseases. The toxemia typical of the wilts is also less common. Progressive killing of cells after invasion, or just before invasion, followed by oxidative breakdown possibly due primarily to the host-cell enzyme system, results in relatively rapid killing, dehydration, and discoloration of the tissue.

Cunningham (24) made a study of the host-parasite relations of a large number of leaf spots. He paid particular attention to the marginal zone between healthy and diseased tissue. The diseases studied were placed in two groups. In one group, represented by shot hole of *Prunus* (*Coccomyces* spp.) and beet leaf spot (*Cercospora beticola* Sacc.), the zone between the dead collapsed cells of the leaf spot and the healthy cells is characterized by mature cells which have become meristematic and solidly packed without intercellular spaces (Fig. 186). Various degrees of cork-cambium activity may be found in this zone and a certain degree of cell-wall suberization. The fungus hyphae are not present in this zone or beyond but may be prevalent in the inner area of the spot where cells have collapsed. It is in this group that "shot-hole" symptoms develop, since the meristematic zone may become a cicatrice and function as an abscission layer, facilitating the dropping out of the center of the spot.

In the second group of leaf spots no cicatrice is formed. The necrotic tissue is collapsed and discolored, and there is a transition zone, sometimes prominent and sometimes barely perceptible and often varying with environment, in which the fungus may be found to a limited extent and in

which various stages of host-cell decline are to be found (Fig. 187). In some cases the fungus grows beyond the necrotic spot to the extent of producing fruiting bodies before macroscopic signs of decline occur, *e.g.*, in celery late blight. In some cases the spot enlarges rapidly until an entire leaflet collapses, *e.g.*, in potato late blight. In other cases the necrotic spot and the fungus are limited by a definite margin, but diffusing toxic materials induce chlorosis, early abscission, and leaf casting, *e.g.*, in early blight of tomato (Fig. 187). In seedling blight of corn (*Gibberella*

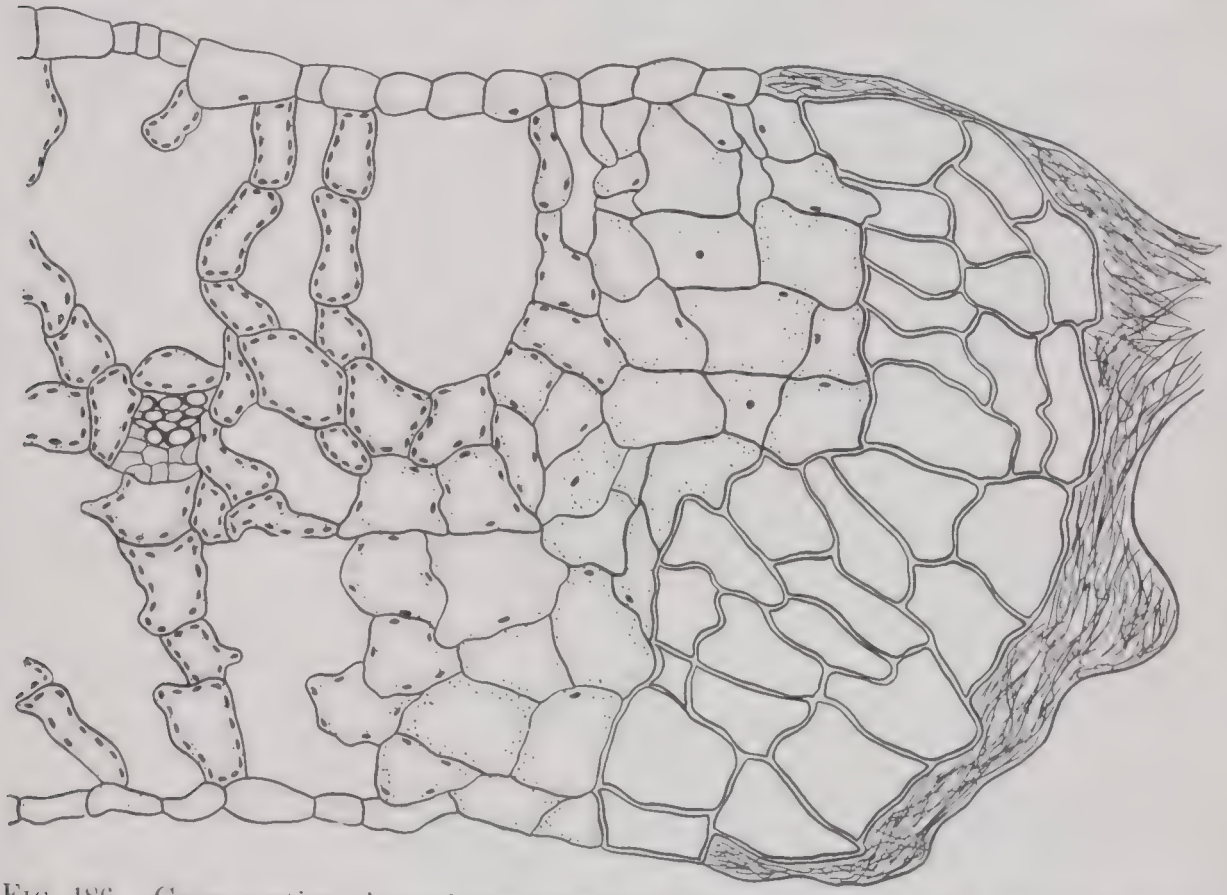

FIG. 186. Cross section through the edge of a lesion incited by *Cercospora beticola* Sacc. in leaf of beet. Healthy tissue is at the left; collapsed portion of the lesion is at the right. At the margin renewed meristematic activity of mesophyll cells has resulted in occlusion of the intercellular spaces and in the formation of a definite cicatrice (compare with Fig. 187). (*After Cunningham.*)

zeae), Pearson (72) found that dark-staining material appeared in the walls and intercellular spaces of the host tissue in advance of the fungus. The fungus is at first intercellular, while at two or three cells at the rear of the advancing hyphae the mycelium becomes intracellular. Zogg (95), in a study of leaf blight (*Pyrenophora calvescens*) of poppy, found that the fungus releases toxic materials as it ramifies through the intercellular spaces, enabling it to assimilate required nutrients from the tissue. At 18 to 21°C. the host responds by the formation of a broad zone of yellow-green cells, but at its optimum (27 to 29°) the fungus outstrips the diffusion of

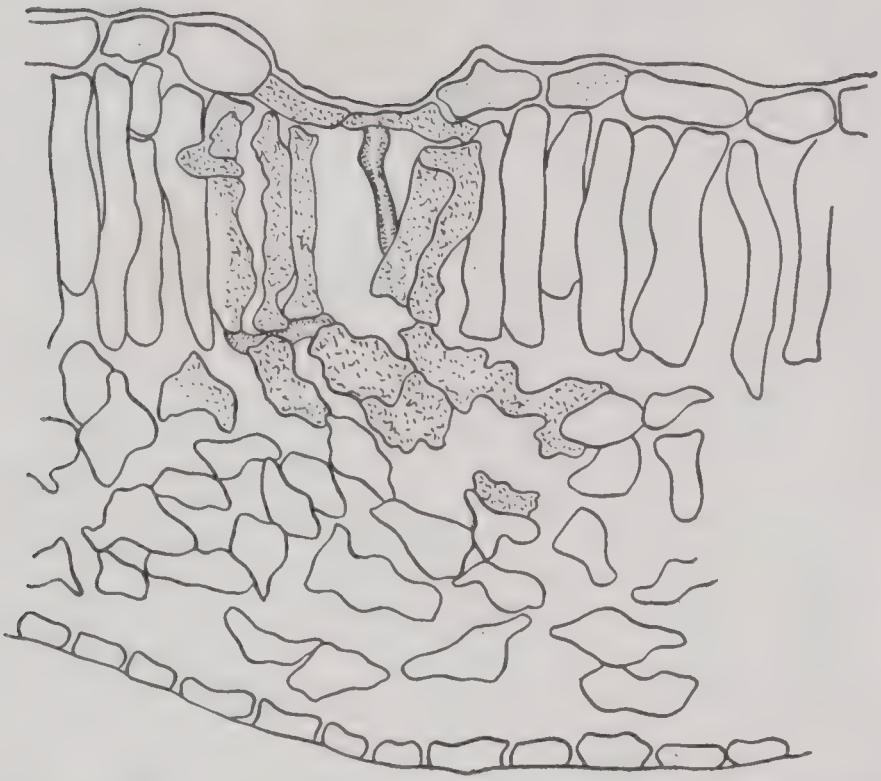

FIG. 187. Cross section through a young lesion on potato leaflet incited by *Alternaria solani*. After direct penetration the mycelium causes progressive necrosis of host cells without any evidence of meristematic reaction of host cells (compare with Fig. 186). (*After Rands.*)

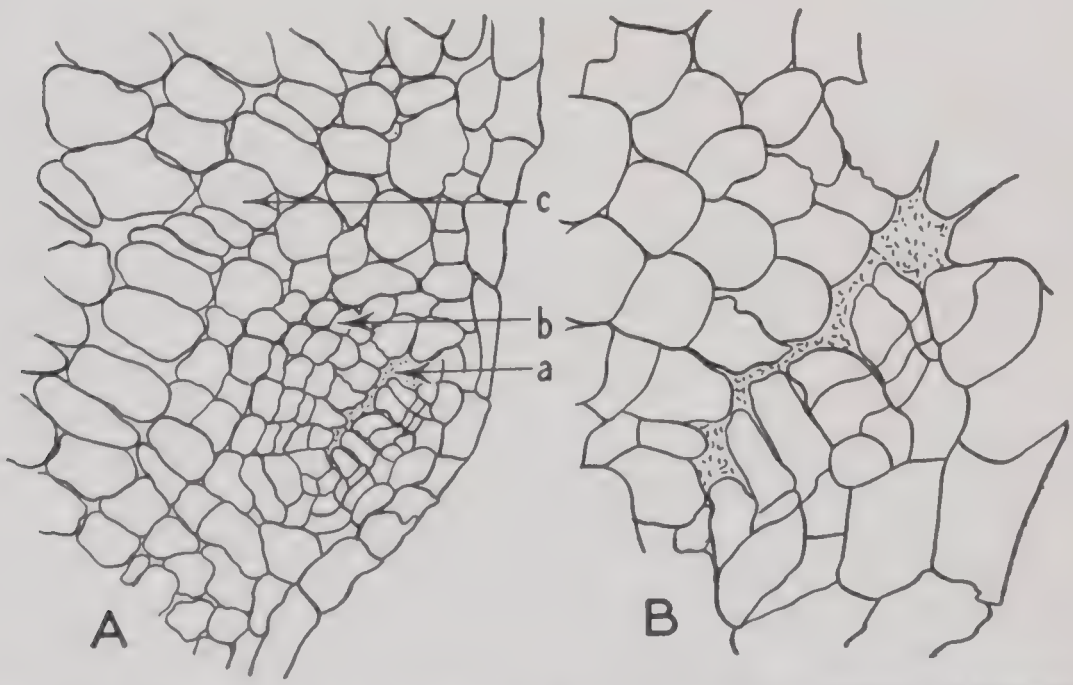

FIG. 188. Reaction of the host tissue to the crown-gall bacterium (*Agrobacterium tumefaciens*). *A*, cross section of an 8-day-old tomato gall. The intercellular spaces (*a*) are filled with bacteria and surrounded by newly formed regions of hyperplasia (*b*) and hypertrophy (*c*). *B*, an enlargement of a portion of *A*. (*After Riker.*)

toxic materials, and the formation of the zone is suppressed. Below 16° and above 30° the fungus grows very slowly, and the host reacts by the formation of a zone of 10 to 30 cells in which gummy substances are formed.

Cellular Stimulation. In many of the examples already discussed there are transient evidences of cell hypertrophy and hyperplasia, often along with cytolysis and necrosis. Cellular stimulation is not patent to any stage of parasitism, but when it is the predominenat feature of pathogenesis, it deserves consideration as a special class. Host plants react by hypertrophy and hyperplasia to a variety of stimuli including chemical agents, mechanical injuries, insects, nematodes, bacteria, fungi, and viruses. Since two of the best known examples, clubroot and crown gall, have already been described in some detail, it is not necessary to repeat here (Fig. 188). It is important to note that, in contrast to other host-parasite interactions, this one is such that growth is stimulated and in the main unregulated. As to how this disturbance of the host is brought about we know little.

Facultative Saprophytism. In most of the cases cited so far the organisms are all capable of saprophytism and may, in fact, live indefinitely as saprophytes. Their attack on their hosts consists in rotting, necrosis, wilting, and in one way or another killing or weakening of the host tissue in advance by means of their excretory products. While the line of demarcation between facultative parasites and facultative saprophytes (p. 9) is not a sharp one it is nevertheless true that groups in the latter class, such as the smut organisms (p. 372), are for the most part parasites with a relatively short saprophytic stage, if any, in nature. With this comes a higher type of parasitism, which, as in the obligate parasites treated in the next section, is essential, since the organism can survive and multiply successfully only in proportion to the extent to which it permits the host to live, grow, and serve as a substrate. The enzymic and lethal effects of the smuts on their hosts are not striking. The fungus for the most part is intercellular without any well-developed haustorial system like that in the obligate parasites, deriving nutrient from the host by absorbing from its cells. Often gaining access to the embryo, the smut organism becomes essentially systemic and grows with the host. It takes over a lethal aspect only when the fungus has sporulated and the host is about ready to mature or at least is no longer useful to it.

Obligate Parasitism. In the above-mentioned types of host-parasite relations following penetration, the organisms are for the most part facultative parasites or facultative saprophytes. We come now to the obligate parasites, where the host tissue must be parasitized to complete the life cycle of the organism. We may consider here chiefly some of the groups which are entirely in this class of parasitism, *e.g.*, the Peronosporaceae in

the Phycomycetes, the Erysiphaceae in the Ascomycetes, and the Uredinales in the Basidiomycetes. Typical cases of each group have already been described in preceding pages. We can only summarize here some of the unique host-parasite relations involved. First is the intercellular mycelium in the Peronosporaceae and Uredinales, which is provided with intracellular haustoria. Various forms of haustoria are to be found, but they all have in common the fact that, when the host is congenial, a phase of symbiotic relation between haustorium and host-cell protoplast is set up at first with only a gradual decline of the latter and collapse only when the spores of the parasite have formed. Whether haustoria invade the protoplast or merely invaginate the plasma membrane is a question still not fully answered. The variations from this congenial relation when resistant hosts are concerned will be discussed later. Thatcher (83) has shown that the permeability of invaded cells as well as of nearby uninvaded cells is altered. Increase in transpiration of plants infected by rust fungi is a common observation. In the Erysiphaceae the same type of haustorial relation occurs, while the hyphae, are, except in a very few species, extramatrical. It is interesting to note that in the powdery mildews and the rusts various degrees of chlorosis occur in the infected region, but some of these cells may show signs of reactivation, and at times zones of green tissue may be retained or reappear.

In the obligately parasitic members of the Plasmodiophorales (clubroot) and chytrids (black wart), the thallus is entirely intracellular, and there is for a time a degree of stimulation of the host cell and finally a decline of the invaded protoplast but not until after considerable hypertrophy and hyperplasia have occurred in invaded and in contiguous uninvaded cells.

HOST RESISTANCE

With some of the common examples of penetration and host-parasite relations before us, we have a background upon which to consider the means whereby the host, particularly varieties and individuals within each species, offers resistance to the pathogens which, under favorable environment, invade or try to invade it. Just as we found the means of invasion and infection to extend over a wide range of cases, so we shall find that resistance takes many forms. Much of the true nature of resistance is not understood. In the main we have some knowledge of alteration in host-parasite relations obtained by histological and cytological methods, but we have only a smattering of knowledge of the physiological and biochemical factors underlying the morphological expressions.

Many schemes of classification of disease resistance have been offered. Cobb (22), working on wheat in Australia in the last part of the nineteenth century, and Orton (69), working on cotton, watermelon, and cowpea wilts

in the southeastern United States early in the twentieth century, pointed out several possible categories. Orton set up three over-all classes, *i.e.*, (*a*) disease escape; (*b*) disease endurance; (*c*) disease resistance. Under disease escape he included cases where the plant avoided infection because of one or another type of superficial character such as glaucous surfaces of foliage, which tended to discourage penetration. Under disease endurance were cases where infection occurred but the host in some way withstood attack sufficiently to grow and produce more satisfactorily than a more susceptible variety. Disease resistance included what might be defined as a high type of resistance: no infection occurred, or if it did occur, the progress of the pathogen was decidedly limited. With more information on the subject than that in the hands of Orton and Cobb it is possible to revise this scheme somewhat and define certain classes a little more rigidly. In this connection the classification of Gäumann (35) is also of interest.

Disease Escape

Apparent resistance to a disease on the part of one variety as compared with another is sometimes due to the fact that for some reason the variety concerned ordinarily escapes the pathogen because of certain environal influences. This is usually a matter of timing of the growth period of the host with the building up of inoculum and the prevalence of environment favorable to infection. In some areas in the midwestern part of the United States early potato varieties escape late blight, because they usually mature before weather favorable to the disease sets in, while later varieties appear to be more susceptible because they are usually in the midst of their growing period when epidemics occur. In another region the same varieties may all succumb to blight, or early varieties may be more seriously affected than late ones.

When the host has a relatively short period of susceptibility, the timing of the period in relation to inoculum and favorable environment may vary with variety. A case in point is the bacterial blight of walnut, *Xanthomonas juglandis* (Pierce) Dowson. Some varieties of walnut are seldom affected seriously in California but are very severely attacked in Oregon. Miller and Bollen (65) consider this a matter of escape. The varieties concerned are late in coming into leaf and exposing vulnerable tissue. In California this lateness results in the new leaves escaping the rainy period, which is essential for infection. In Oregon, on the contrary, weather favorable to infection is more protracted and extends beyond the time when leaf tissue is exposed.

Many records of apparent differences in resistance of varieties of a species to a given disease may be based on escape of the type illustrated above. While this is really not resistance at all, such average behavior of a variety

in a given locality may make it particularly valuable there. In many cases early varieties are used because they escape one or another disease. Or varieties may be chosen because they can be planted later than others, with the advantage that they escape a set of conditions favorable for a given disease. It is in the use of such varieties in another environment that their limitations are found, especially when evaluations are made on an empirical basis without a clear understanding of the underlying pathological factors involved.

Exclusion of the Pathogen

There are numerous cases in which the fundamental basis of resistance in the variety concerned lies in characters of the plant which preclude or greatly reduce the chances of infection. This type of resistance is manifested through the interaction of host and parasite as penetration takes place. When the barrier of exclusion is overcome by artificial or natural means and infection accomplished, the variety concerned shows the same type of susceptibility as varieties not endowed with this property. The nature of this type of exclusion is not always well understood, and the line of distinction between it and escape is not definite. Two well-defined subtypes will be discussed next.

Mechanical Exclusion. It is not uncommon for parts of the host plant to pass from a period of susceptibility into one of resistance or vice versa. This is sometimes the result of resistance due to some character at the threshhold. A pertinent example is the case of onion in relation to the smut organism (p. 392). The cotyledon of common onion is susceptible to infection up to the time of midgrowth. The cotyledon of the Welsh onion, however, is susceptible only when very young, and since it becomes resistant rapidly, it serves to protect the plant from systemic infection. Evans (29, 30), in a study of the relation of parasite to host, indicates that resistance is in part intracellular but to some extent is contained in the nature of the outer membrane. In any case the rapid development of resistance in the protective cotyledon of Welsh onion makes it a resistant species, while the common onion is a very susceptible one. Resistance in corn to common smut is due in part to protection of vulnerable node tissue by sheaths which have passed into a resistant stage. Varieties with poorly protected nodes are the more susceptible when epidemic conditions prevail. Rosenbaum and Sando (77) studied the relation of the age of tomato fruit to infection by the nailhead fungus, *Alternaria tomato* (Cooke) G. F. Weber. In this case the immature fruits are infected by direct penetration of the fungus. As the fruit grows, it becomes less liable to infection, and at maturity it is nearly immune. It was shown that a fairly close correlation existed between increase in resistance of the cuticle to mechanical puncture

and increase in resistance to infection. Curtis (25) pointed out that the resistance of some varieties of plum to brown rot is due in part to the relative thickness of the cuticle. In some varieties patches of the fruit surface occur with little or no cuticle, and these are more liable to direct attack than those which become uniformly cuticularized early.

Numerous cases are found in older literature in which size of stomata and relative glaucousness of the surface of leaf and stem were factors affecting resistance of varieties to one or another pathogen (88). Some claims have not been substantiated by further inquiry, and, by and large, these factors are now regarded as of little importance.

When the pathogen penetrates through stomata and requires that stomata be open before invasion can occur, the time of diurnal opening and closing of the aperture is important. In some of the rust fungi penetration may occur through open or closed stomata. In other cases, as in *Puccinia graminis*, invasion is most successful if stomata are open. Hart (41) has shown that in some varieties of wheat which are resistant to many races, *e.g.*, Hope, the stomata are slow to open in the morning, and thus the fungus is largely excluded during the early morning hours when dew is still present and is favoring infection in varieties whose stomata open much earlier. This particular type of resistance has been called functional resistance.

Chemical Exclusion. There are relatively few cases of exclusion of the parasite by chemical exudate from the host. Brown (11) showed that electrolytes diffused from the host cell through the cuticle into the infection drop and stimulated or retarded the fungi present, depending upon the nature and concentration of the material. The most clear-cut case of exclusion of a pathogen by chemical means is that of onion smudge. Here the fungus normally invades the dry outer-scale tissue of the maturing bulb, establishing itself saprophytically and then penetrating the cuticle of the underlying fleshy scale directly (86, 87). The usual freedom from infection of outer onion scales of colored varieties has been observed widely. It was shown that phenolic compounds, protocatechuic acid (2) and catechol (62), are intimately associated with the pigments in the outer scales. Moreover they are water-soluble, colorless, and toxic to fungi. As the infection drop comes in contact with the outer dry scale, which consists of dead tissue, the toxic phenols become soluble, diffuse into the infection drop, and kill the fungus before infection can occur. Thus the resistant character associated with scale pigment is one in which the fungus is incapacitated before infection. To a lesser degree the same material functions in restricting infection of the bulb necks by the neck-rot fungi (*Botrytis* spp.), but the opportunity for effective exclusion is not as great as with smudge (44). Ramsey (74) has shown a similar relation between phenols and resistance with the *Diplodia* bulb rot of onion (*Diplodia natalensis*

P. Evans). Campanile (19) has shown resistance of red garlic to *Helminthosporium allii* Campanile to be based in part upon the same phenomenon.

It has been demonstrated that other materials toxic to pathogens exist in the fleshy scales of onion (44, 70, 86, 87). These come into play after invasion and will be discussed in the next section.

Resistance Based on Internal Factors

In a large majority of the cases, resistance to disease in plants consists in interactions which take place after penetration. As already pointed out, many cases of penetration, direct and through stomata, occur without infection taking place. There does not appear to be any connection between the attraction or stimulus to penetrate and the state of affairs which permits pathogenicity. What it is that determines whether certain parasites select certain hosts and not others, while other parasites have little specialization and are omnivorous, no one has as yet defined. Whether so-called resistance is more often actually a matter of retardation by the host or purely a lack of attraction on the part of the host is still a moot question. In the main, however, if a given pathogenic species is adapted or accustomed to attacking a host species, it usually goes through the initial stages with all varieties or individuals of the species. In resistant individuals or varieties the usual chain of events is upset in some manner and degree. The manifestations of resistant reactions are many. For the most part we know about them by a study of the histological and cytological pictures. While morphological reactions are sometimes specific and suggestive, they do not reveal the physiological reactions which underlie them. The biochemical and physiological reactions are still more elusive, and here particularly we find the basic questions still largely in the realm of the unknown.

Morphological Manifestations of Resistance. Once the parasite has penetrated the resistant host, deviation from the usual reaction of the susceptible host appears. There may be merely a paucity of penetration and decided limitation of ingress beyond penetration without any distinctive morphological reaction. This is the usual case in type A resistance in cabbage to the yellows organism (p. 253). In other cases there are more or less characteristic morphologic responses in the form of alteration of cell walls, *e.g.*, thickening, suberization, cambium activity, or cell hypertrophy.

In tobacco root rot, *Thielaviopsis basicola* (Berk.) Ferr., there are many resistant varieties which have been developed through breeding. The pathogen is concerned primarily with the cortex and periderm of the roots. Invasion of roots of resistant plants takes place commonly, but the degree of infection is decidedly more limited than in susceptible varieties. The relation of soil temperature to development of the disease is discussed

elsewhere (p. 548). One of the most common points of penetration is at the ruptures of the root cortex by secondary roots. Conant (23) compared resistant and susceptible varieties in this regard and found that at soil temperatures favorable to the disease there was decidedly prompter cork-cambium formation in the parenchyma tissue of the root around secondary root ruptures. The fungus was definitely retarded or excluded by the cork cambium. At high soil temperatures, unfavorable to the disease, susceptible varieties showed greater cambium activity than at lower temperatures. He reported that the initial penetration of the fungus seemed to stimulate the cork-cambium activity in resistant roots. In later work on this subject Jewett (51), growing plants in nutrient solutions, failed to corroborate Conant's finding as to correlation of cambium activity with resistance and believed the latter to be based on chemical differences rather than on anatomical factors.

The tubers of the potato variety McCormick are more resistant to decay incited by *Pythium debaryanum* than are other commonly used American varieties. Hawkins and Harvey (45) studied the progress of the fungus in tissue of resistant and susceptible varieties. The intracellular fungus was considered by them to pass from cell to cell, primarily by mechanical pressure upon the cell wall. They noted that the mycelium seemed to be impeded in this puncturing process by the walls of cells of resistant tubers. This apparent resistance was correlated with the resistance of the membranes to mechanical pressure. The solvent action of the fungus on walls of the host cell has not been investigated critically, however. Bonde *et al.* (7) are of the opinion that the tuber-rot resistance to late blight in potato varieties they studied is due in part to the thickness of the periderm. Löhnis (63) came to the conclusion that, while rot resistance in the Dutch variety Bravo was intimately connected with the cork cambium, there were no anatomical characters in this meristematic layer which were characteristic of the resistant variety.

It was pointed out with black stem rust of wheat (p. 401) that a type of resistance known as mature-plant resistance is characteristic of some varieties, *e.g.*, Hope, and is not necessarily correlated with resistance associated with the intracellular reactions of host and pathogen upon which differentiation of physiologic races is based. Mature-plant resistance from the standpoint of host anatomy has been studied critically by Hart (42). The rust fungus attacks thin-walled parenchyma tissue, in which intercellular mycelium develops, and haustoria are formed abundantly in susceptible hosts. In varieties which are mature-plant-resistant, however, the relative amount of thin-walled parenchyma tissue in relation to thick-walled sclerenchyma decreases as the plant approaches maturity. Thus in a rust epidemic which builds up after midseason, which is the form most

destructive to the crop, varieties having this type of anatomical organization show marked resistance.

Intracellular Manifestations of Resistance. It is a common observation that the relation of the dicaryotic phase of rust fungi to the host is made manifest by symptoms which are characteristic of the particular variety of the host and race of the pathogen concerned. Taking black stem rust as an example, for a given race a series of varieties may usually be found, some of which are susceptible, giving a 3 or 4 reaction, while others have definite signs of resistance as shown by 0, 1, or 2 reactions. The relation of the pathogen to resistant and susceptible hosts has been studied most critically by Allen (1). She studied comparatively Baart, a variety of wheat susceptible to races 3 and 19; Kanred, a variety susceptible to race 3 and immune to race 19; and Mindum, which is immune to race 3 and resistant to race 19. In Baart and Kanred, race 3 forms normal haustoria, and the host cells in proximity to the fungus are stimulated to increased metabolic activity, while the host nuclei increase in volume. The stimulation of cells outlying the infected cells appears sooner in Kanred than in Baart. Plastids are decreased in size until increased metabolic activity seems to bring a check in this effect. The host nuclei eventually collapse. Race 3 also forms haustoria in immune Mindum, but the living contents of the cell flow rapidly to the haustoria, condense around them, and the whole host cell promptly collapses. The fungus haustorium dies a little later. As haustoria form and host cells collapse, the protoplasm of the intercellular hyphae moves to the haustoria. The fungus is thus impoverished, while the outlying host cells become plasmolyzed. When Kanred, immune to race 19, was inoculated with the latter, essentially the same reaction occurred as that between Mindum and race 3. This picture with some variation is found in most rusts studied with reference to seedling resistance as opposed to mature-plant resistance. It is clear that there is no barrier to penetration of immune hosts, but there is a pronounced incompatibility between the immune host cell and the pathogen. Whereas a more or less congenial relation exists during the initial stage of infection of the susceptible host, permitting sporulation of the fungus, this is not the case with the immune host. It is still a matter of speculation whether the fungus produces a material toxic to the host cell or whether something in the host cell is toxic to the fungus. In any case, when one considers the large number of races of *Puccinia graminis*, each with its specific relations to various hosts, it is evident that any toxic materials, whether they are metabolites of one organism or the other, must be highly specific.

Many researches in the past have been directed toward demonstrating within resistant tissues substances which would account for resistance by being toxic to one or another pathogen. Much of the earlier literature

has been reviewed (88). In general it may be stated that little evidence has been brought to bear on the numerous cases which have been explored. The chemical exclusion of organisms by soluble phenols in dead outer onion tissue has been discussed in an earlier section. It was pointed out that from the fleshy onion tissue there arise, on crushing the tissue, volatile materials, probably allyl sulfides, which are extremely toxic to onion pathogens as well as other fungi. In spite of the presence of this material in the fleshy scale, the smudge organism will invade white and colored bulbs if the inoculum is placed on the fleshy scale after the dead outer scale is removed. Here we have the paradox that the fungus prefers this tissue in spite of the sulfides, which are toxic, and the color substances, which are present in the outer epidermal cells of the fleshy colored scale. It is well to emphasize, however, the importance of the relation of the fungus to the toxic substances. When the inoculum contacts dead colored tissue, soluble substances may diffuse out readily; when it contacts fleshy scales, the cuticle, a relatively thick cellulose wall, and a plasma membrane stand between the fungus and the host protoplast. Moreover, as the fungus punctures the cuticle, it first dissolves part of the cellulose membrane (Fig. 184), and while this is going on, the pigment disappears and the protoplasm shows signs of decline. Thus the fungus metabolites and products of wall digestion may diffuse in advance and disarm the host cell by upsetting its defensive mechanism. In spite of this it has been shown by Hatfield *et al.* (44) and by Owen *et al.* (70) that the volatile toxic materials in onion have a bearing on the pathogenicity of both the smudge and the neck-rot organisms.

The case cited in the onion is illustrative of the complexities which arise in attempts to delineate any substance or class of substances in the host cell which make for resistance. The fact that materials toxic to a given organism exist in a given variety of host is in itself no proof that these materials are responsible for resistance. More lines of evidence must be had to prove the case.

As an example of misleading facts, the case of volatile sulfur compounds in crucifers in relation to clubroot may be cited. It was claimed that black mustard was resistant to clubroot because of the toxic effect of the allyl isothiocyanate present in that species (76). It was shown that this substance, highly volatile, is very toxic to many fungi including the clubroot organism (50). It occurs in the host, however, as a glucoside, sinigrin. When this substance was prepared, it was found to have no toxic effect whatsoever (90). It was still possible that a fungus entering the cell might set up enzyme activities which would break down sinigrin and release the toxic material, which in turn would incapacitate the fungus. In the meantime an analysis of strains of black mustard revealed that some were

naturally resistant and some naturally susceptible to clubroot (89). Analysis of the two types for allyl isothicyanate and the equally toxic phenethyl isothiocyanate occurring in the roots showed no difference between resistant and susceptible strains (80). In another line of approach, resistant and susceptible plants were grown in sand with nutrients devoid of sulfur (73). This resulted in plants without detectible isothiocyanates. However, the sulfur-free resistant plants were still resistant. Thus it was shown by various lines of evidence that the very toxic material in the tissue had nothing to do with its resistance.

The approach to the nature of disease resistance on the basis of presence or lack of nutrients essential to the pathogen has not been taken by many investigators, but it is one which deserves much more consideration. The study of Vasudeva (85) is illustrative. He found that low nitrogen depresses the excretion of propectinase by *Botrytis allii*, which is normally nonpathogenic on apple-fruit tissue. However, if a trace of soluble nitrogen is provided in the inoculum when this fungus is applied to apple-fruit tissue, it becomes distinctly pathogenic. The work of Dickson cited under seedling blight of cereals (p. 342) illustrates the importance of cell-membrane constitution in relation to resistance and susceptibility to *Gibberella zeae* at different soil temperatures.

VARIABILITY IN PATHOGENICITY OF MICROORGANISMS

The first clear evidence that there exist, within a given morphologic species of microorganism, strains which differ in pathogenicity was that presented by Eriksson (28) in a series of papers on *Puccinia graminis* Pers. and other rusts in the early nineties. He showed, first with black stem rust, that the organism collected from wheat did not infect rye, oats, and certain other hosts, while collections from other host plants showed other ranges of pathogenicity. These so-called strains were practically identical in morphology and infected the same alternate host, but they differed in their physiological characters as expressed in their specific pathogenic properties. A few years later Ward (93, 94) in England studied the brome rusts and found a similar phenomenon in specialization of strains upon certain species of *Bromus*. Ward recognized that there were certain host species which were infected by more than one strain, some strains being more virulent than others on such an intermediate host. He believed that he had sufficient evidence to show that, when a given strain was grown for a sufficient number of vegetative generations on a noncongenial host, its pathogenicity was changed to the extent that it would infect a third, previously completely resistant, host species. This intermediate host species was designated by Ward as a bridging host. This theory, known as the **bridging-host theory,** assumes a certain plasticity of the pathogen, which

is influenced by the host substrate in such a way as to change its selective pathogenicity. The theory was tested critically in the United States with *P. graminis* by Stakman (81) and found not to hold. In general, Ward's theory of bridging hosts has been discarded by later workers in so far as rusts are concerned.

In 1911, Barrus (3, 4) reported a study of resistance of bean varieties to different collections of the anthracnose organism, *Colletotrichum lindemuthianum* (Sacc. & Magn.) Briosi & Cav. He differentiated two strains of the organism on the basis of reaction of host varieties. Certain varieties were highly resistant to one strain and very susceptible to the other. Other varieties showed the opposite reaction. Barrus designated these strains as *alpha* and *beta*. Burkholder (18) later described a third strain *gamma*. This is the first instance on record in which distinct physiologic races within a morphologic species of a pathogen were defined on the basis of horticultural varieties of a single host species rather than on the basis of host species or genera as had been done previously in the case of the rusts.

In 1917, Stakman and Piemeisel (82) and, in the following year, Melchers and Parker (64) reported from independent investigations another significant fact. They found that collections of *P. graminis tritici* from durum wheat (*Triticum durum* L.) and from bread wheat (*T. vulgare* L.) were not alike pathogenetically. This was the first evidence that the varieties of Eriksson were not pathogenetically homogeneous. Further work by Stakman and associates rapidly built up evidence that many strains exist within each of the subspecies of *P. graminis*.

Since the work of Eriksson the strains which differ pathogenetically within a morphologic species have been called by various names, *e.g.*, specialized form, specialized race, biologic form, biologic race, biologic species, physiologic form, physiologic race. By action of the International Botanical Congress at Amsterdam in 1935 the term **physiologic race** was adopted. The definition as presented in the Proceedings is as follows:

> It is recommended that the term "physiologic race" be substituted for "physiologic form" as the former seems more appropriate. It is recommended further that the word "race" be used in general to designate biotypes or groups of biotypes that differ from each other in physiologic characters.

Variability in fungi and bacteria has now become generally recognized, and a number of reviews of the literature are available (20, 55, 60, 75). The study of genetics of the fungi has shown that variability in these organisms, as in higher plants, may be genetic or environmental. The vegetative phase of the fungi for the most part is haploid in Phycomycetes, Ascomycetes, and Fungi Imperfecti. In the Basidiomycetes it is for the most part dicaryotic, in which mated haploid nuclei divide conjugately.

Where fungi are heterothallic, it is possible to mate compatible thalli and study the inheritance of gene-controlled characters. By this means it has been shown that many characters are monogenic and that gene mutations occur. In a few cases it has been shown that pathogenicity is also gene-controlled. In the apple-scab organism, as stated earlier, at least two pathogenic characters have been shown to be controlled by two distinct gene pairs. In races of the wheat-bunt organism it has been shown that pathogenicity is inherited, and probably it is in this case polygenic (49). New races of oat smut have been produced by hybridization (48). As already shown in the discussion of black stem rust, the pathogenic characters of the various races are gene-controlled.

In the Basidiomycetes the effect of complementary genoms on the dicaryote has an important effect upon pathogenicity. Buller (17) pointed out that even though the two haploid nuclei did not fuse in the thallus, the dominant gene in one of a pair had the same effect on the thallus as if the two haploid nuclei had fused. It is shown further by Johnson and Newton (55) that a so-called race of *P. graminis*, as determined by host reaction, may consist of various combinations of haploid nuclei which are genotypically different but happen to have in combination the same phenotypic effect on the thallus. This leads to the consideration of heterocaryosis (39), which is a term applied to a state in which nuclei of more than one genetic type occur in the cell of the thallus. In some of the Fungi Imperfecti and Ascomycetes and in all of the Phycomycetes the thallus is multinucleate. If these nuclei are of different genetic constitution, they may affect the pathogenicity of the thallus differently. In many Fungi Imperfecti and Ascomycetes anastamosing of hyphae and intermingling of nuclei may take place. Thus heterocaryosis of fungi which do not have a sexual stage may be another basis of variation in pathogenicity of microorganisms.

A third basis of variability is gene mutation. That such occurs in the fungi is no longer questioned, and many mutations have been recorded in *Neurospora* and some in *Venturia*. If mutation takes place in a cell of a uninucleate thallus like that of *Venturia* or *Penicillium*, the new strain is likely to be distinct and remain so in repeated transfers. When a mutation occurs in a gene in a nucleus of a multinucleate thallus like that of *Botrytis*, the opportunity for its expression on the thallus is much less, since the dominance of the genes in the other nuclei in the cell may be such as to prevent it. By chance, through later anastamosis, the mutant nuclei may predominate in a cell, and the effect on the thallus may appear. Further anastamosis may result in more dominant nuclei in a cell, and reversion to the parent type follows (71).

In summary then it may be said that, from the standpoint of pathogenicity in the fungi, the character is definitely controlled by genes. It may

be monogenic or polygenic, and many distinct genes may occur which control distinct pathogenic capacities for different host varieties. Variability of a fungus may result from fusion of dissimilar haploid nuclei and segregation of pathogenic genes in the meiotic division of the diploid nucleus. It may result from mutation in the haploid thallus, and it may be the result of change in the nuclear population of multinucleate thalli through anastamosis of hyphae and intermingling of haploid nuclei.

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CHAPTER 15

DISEASE CONTROL THROUGH EXCLUSION AND ERADICATION

Plant diseases affect the lives and well-being of so many individuals that the most popular interest in them concerns the means by which they may be prevented. The solution of control problems is the ultimate purpose of a distinct science of plant pathology. As in the sciences concerned with the pathology of man and of animals, many control practices for plant diseases have originated by empirical cut-and-try methods long before the true nature of the diseases concerned was understood. Even today much testing of method and material is carried out in a hit-and-miss fashion, when a more intelligent approach could be made through a study of (*a*) the nature of the disease; (*b*) the variability, life history, and environal response of the causal agent; (*c*) the relation of environment to the predisposition of the host and to host-parasite interaction; (*d*) the defense reactions of the host, their response to environment, and the nature of their inheritance. The real function of plant pathology is to provide a fundamental basis upon which scientific rather than empirical control procedures may be established.

In this and in succeeding chapters the various procedures for control will be discussed. In so far as nonparasitic diseases are concerned, the regulation of environment, where feasible, or the control of nutrition of the host makes up the chief approach. When a microorganism or an infectious virus, and ofttimes a biological vector, is added to the disease causation complex, the approach must be modified. This may take the line of eradication of, avoidance of, or protection against the pathogen. It may become a matter of fortifying the host against the infective agent by certain cultural practices. It may be a process of immunization of the host by development of resistant strains through hybridization and/or selection. It may be protection through the use of fungicides.

Control measures may be divided into two major groups, prophylaxis and immunization. **Prophylaxis** implies the protection of the host from exposure to the pathogen, from infection, or from the environal factors favorable to disease development. **Immunization** refers to improvement of resistance of the host to infection and to disease development. It is commonly referred to in plant pathology as **disease resistance.** In general, it is used as a means of control by the development of strains of the host, through hybridization and or selection, which are more resistant to one or more pathogens, viruses, or environal complexes. Acquired immunity comparable to, but not analogous with, that used widely with animals is possible so far only with virus diseases. Temporary non-heritable resis-

tance is sometimes provided through regulation of predisposing and post-infection environal factors such as temperature, soil moisture, and host nutrition. Prophylaxis is applied to a wide variety of control measures. They may be considered under three subgroups: (*a*) exclusion, (*b*) eradication, (*c*) direct protection. Under **exclusion,** measures are designed to keep the pathogen from entering the sphere in which the host is growing or to reduce to a minimum the extent to which the pathogen is introduced. These measures include compulsory quarantines and inspection; embargoes; voluntary inspection and certification of planting stocks, together with their production in areas where the pathogen is minimized or excluded by unfavorable environment; inspection of perishable fruits and vegetables at shipping points. **Eradication** consists in measures which are concerned with the elimination of the pathogen after it has become established in the sphere where the host is growing. These measures include eradication of alternate hosts; eradication of wild or other overwintering hosts; eradication of overwintering debris as sources of primary inoculum by rotation, sterilization, and or sanitation; eradication of the pathogen from propagative parts of the host such as seeds, tubers, and bulbs; eradication from growing parts of the host by fungicides after infection has occurred. **Direct protection** refers to measures in which it is assumed that the host will be exposed and that infection will occur unless the procedures are undertaken. Most spraying and dusting of foliage is direct protection. Some seed treatments are eradicant; others are protective. Some of the other direct protective measures in common use are regulation of environment, as in greenhouses and in storage; timing of planting, cultivating, and harvesting to favor the host and discourage the pathogen; harvesting and handling practices, precooling, prestorage curing; protection from frost, suboxidation, heat, and ray injuries; correction of mineral deficiencies.

EXCLUSION

The clarification of the pathogenic nature of fungus and bacterial organisms in the middle of the nineteenth century soon focused attention upon the long-distance dissemination of such organisms. Before the middle of the century the powdery-mildew disease of grape, *Uncinula necator* (Schw.) Burr., had appeared in Europe, and although the true nature of its cause was still debatable, it was regarded by vineyardists as having been introduced with nursery stock from the United States. The growing importance of the phylloxera gall louse (*Phylloxera vitifoliae* Fitch) about 1870 instigated the use of the more resistant seedlings from America as root stocks. In the decade of 1870 the rapid development of downy mildew of grape, *Plasmopara viticola* (Berk. & Curt.) Berl. & DeT., after the recent introduction of the pathogen, also from America, was the cause for even greater

alarm. About 1860 onion smut (*Urocystis cepulae* Frost) was discovered in New England. Found later in France, it was at first thought to have been imported from America, but its discovery later in the Black Sea region near the native habitat of the onion indicates that it probably was transported therefrom with the host. The powdery-mildew organism of gooseberry, *Sphaerotheca mors-uvae* (Schw.) Berk. & Curt., was introduced from America into southwest Russia about 1890, and it spread throughout western Europe, appearing in Ireland in 1900. It has become a far more serious disease in Europe than in America. The rust pathogen of snapdragon (*Puccinia antirrhini* Diet. & Holw.) was first noted in California in 1895 and has spread throughout the United States and to Bermuda since that date. It was found in Europe for the first time in northern France in 1931, apparently having been introduced recently from America. Once established, it spread with extreme rapidity, as shown by the fact that it was recorded in England in 1933; in Germany and Denmark in 1934; in Sweden, Czechoslovakia, Switzerland, and Italy in 1935; in Austria, Estonia, Poland, Rumania, and Egypt in 1936; and in Bulgaria and Palestine in 1938. In many of the new locations the disease is very destructive.

Notable examples of relatively recent introduction of foreign pathogens to American shores are white-pine blister rust, *Cronartium ribicola* Fischer, discovered in 1906; chestnut blight, *Endothia parasitica* (Murr.) And. & And., in 1904; citrus canker, *Xanthomonas citri* (Hasse) Dows., in 1912; potato wart, *Synchytrium endobioticum* (Schilb.) Perc., in 1918; wheat flag smut, *Urocystis tritici* Koern., in 1918; and Dutch elm disease, *Ceratostomella ulmi* Buisman, in 1930.

Probably many other pathogens have been introduced without having become established. It is obvious that there is an important distinction between introduction and establishment. Apple scab, *Venturia inaequalis* (Cke.) Wint., and apple bitter rot, *Glomerella cingulata* (Stone.) Spauld. & Schrenk, are not known or are rare in California, although it is generally believed that the pathogens concerned were introduced repeatedly from eastern states on nursery stock before quarantine regulations were established. Black rot of cabbage and cauliflower, *Xanthomonas campestris* (Pam.) Dows., is not known in Pacific coast regions, although the pathogen has undoubtedly been introduced repeatedly in infected seed. Lack of establishment in these instances is explained by unfavorable environment in the region concerned. In some cases introduction, though followed by establishment, does not result in the disease assuming a major role. About 1912, powdery scab of potato, *Spongospora subterranea* (Wallr.) Lagh., became established in some areas in Maine, having been introduced from Europe or Canada. The areas were quarantined for a period of years, and potatoes shipped to market were subjected to inspection. Eventually the

disease was found in other regions of the United States and study indicated that it would probably not assume a major role. The quarantine was then lifted, and the fact that powdery scab remains a rare disease justifies that procedure. In still other cases a disease organism of minor importance in a country where it is endemic has, upon introduction into another region or country, found a strain of host more susceptible to it and an environment more favorable for its development. Such was the case with chestnut blight, as described later in this chapter.

History of Plant Quarantines

The growth of quarantine regulations in the United States and elsewhere has been under way since about 1870. The exclusion of insect pests has been intimately associated with that of fungus and bacterial pathogens in this development. In the United States, plant quarantines were first advanced on the initiative of individual states. Foremost in this movement was California, where the first act was passed in 1881. It was designed to prevent the distribution of the grape gall louse. The law was amended and strengthened during succeeding years with the result that all nursery stock seeking to enter the state from other states or from abroad was thoroughly inspected and disinfected or destroyed as the particular occasion might require. Gradually other states passed quarantine laws and set up organizations to enforce them, until, at present, every state is so equipped. These regulations have been directed primarily toward perennial propagative material in the form of trees, shrubs, and flowering bulbs. Little or no application has been made to edible fruits and vegetables or to seeds of annual plants, all of which, of course, may harbor and distribute pathogens.

While state organizations restricted interstate movement of nursery stock by a system of inspection and certification, the national government took no action in the restriction of importation from abroad until 1912, when the Federal Plant Quarantine Act was passed by Congress. Until that time importation of pathogens was restricted only by the activity of individual states which happened to border upon the oceans or upon the foreign countries to the north and south.

Previous to 1912 restriction of movement of plant pathogens and insects was begun in Europe and Australia. In 1874 the Colorado potato beetle was found established in Germany and by drastic action was eradicated. An embargo against further importation of potatoes was decreed in 1875. The grape gall louse, originally introduced into France about 1859 from the United States, was introduced into Victoria, Australia, from France in 1872 and became established. Drastic legislation directed toward its suppression was passed in 1877. The widespread establishment of the gall louse in Europe and its severe destructiveness in some areas led to an international conference on regulations for its restriction at Berne, Switzerland, in 1881.

Various European countries enacted quarantine legislation directed particularly against American products. One of the most important was the embargo of 1898, which was directed toward the San Jose scale then conspicuous in California, to which state it had been introduced from China about 1880. Various attempts were made to secure Congressional legislation to provide a system of national quarantine for the United States, beginning in 1892. The first act to pass the Congress was the Plant Quarantine Act of 1912.

The Plant Quarantine Act authorized the Secretary of Agriculture by permit to **condition, regulate,** and **restrict** the entry into the United States of nursery stock and other plants and plant products and make it unlawful to import plants and plant products otherwise; it further authorized the Secretary to **prohibit** the entry of nursery stock and other plants and plant products. Various amendments were added in succeeding years. The Act thus provided a means of setting up embargoes and quarantines. It provided that the administration of the Act be assigned to a Federal Horticultural Board to be appointed by the Secretary. The first board consisted of C. L. Marlatt and A. F. Burgess of the Bureau of Entomology, W. A. Orton and Peter Bisset of the Bureau of Plant Industry, and G. B. Sudworth of the Forest Service. The Federal Horticultural Board was succeeded in 1928 by the Plant Quarantine and Control Administration. Since 1933 the enforcement of the Act has been within the organization of the Bureau of Entomology and Plant Quarantine of the U.S. Department of Agriculture. This agency is empowered to promulgate regulations as to the importation of propagative materials and may extend them to all fruits, vegetables, seeds, and any other materials which may be suspected of harboring injurious insects or pathogens. While the Plant Quarantine Act applies also to the interstate movements of such commodities, the regulation of such movements is left to the state enforcement bodies by mutual agreement and arrangement. In general, the movement and sale of nursery stock now requires prior inspection and certification for both intrastate and interstate commerce. Exclusion of propagative materials from abroad is enforced by the national bureau, with provision made for the introduction, under supervision, of new varieties and of materials for scientific experimentation. Certain bulbs may enter under inspection permits. Certain commodities such as potato are excluded from certain countries where potato wart occurs. Fruits, vegetables, and seeds are allowed to enter under permit and subject to inspection at the point of entry.

Factors Affecting the Efficacy of Quarantines

Introduction of a pathogen or virus into a new locality may be brought about in various ways: (*a*) the host may be the carrier; (*b*) inert materials such as packing materials may carry resting stages of the organism; (*c*) in-

sect vectors and birds may transport it; (*d*) air currents may carry the pathogen long distances. The first two modes of distribution lend themselves to curtailment by quarantine measures. The last two, by and large, are beyond the control of man and offer a major limitation to the control of disease by exclusion. Birds have been shown to carry fungus spores, and some species of birds, of course, migrate from one continent to another. However, there is little evidence of important long-range distribution by this means. Insect transmission, in so far as it is now understood, is most likely to be important within relatively small geographic areas. Those pathogens whose spores are readily adapted to dissemination for long distances by air currents, such as the rust fungi and powdery-mildew fungi, are particularly difficult to restrict except by major natural barriers. The most effective natural barriers are the large oceans. Although spores are carried long distances in the upper air, there is as yet little evidence of any measurable amount of transoceanic dissemination by wind. Within continents certain natural barriers exist, the most important being high mountain ranges. While these may serve to slow down or impede the distribution of wind-borne spores, they cannot be regarded as effective against all.

It may be assumed, therefore, that pathogens which are readily air-borne will be eventually disseminated throughout a continent and become established in those areas where susceptible hosts and favorable environment prevail. In fact, such distribution and adjustment has probably taken place within larger continental areas regardless of state lines and international boundaries. It is with regard to pathogens carried with host parts or with nonhost carriers that control through regulation may be effective. It is against such pathogens that quarantine regulations are directed primarily. Many plant pathogens in other continents have been listed which conceivably may be prevented from causing losses to American agriculture, while still others of importance on the American continent, such as the fire-blight organism, can still be excluded from parts of Eurasia and Africa.

At this point we shall consider the pertinent facts relating to a few of the important cases of introduction and establishment of foreign pathogens in the United States since about 1900.

Some Important Introductions of Foreign Pathogens

White-pine Blister Rust. The eastern white pine (*Pinus strobus* L.) is native to northeastern United States and southeastern Canada. It was introduced into Europe during the last century. Nurserymen in Europe engaged in the practice of producing white-pine seedlings often from seed secured in the United States. Many of these were imported into this country for use in the reforestation program. Blister rust was first reported in

the Baltic regions of Russia, supposedly on a native species of pine (*Pinus cembra* L.). The fungus affects only five-needled pines, most of which are susceptible. In 1883 the rust was reported as widespread on the European continent, and it was reported in England in 1892. The alternate hosts of this long-cycle rust are currants and gooseberries (*Ribes* spp.), a large number of species of which are susceptible.

The fungus penetrates the needle of the pine, grows into the bark, and causes cankers which mature after 2 years, on the surface of which are produced sacs or pustules containing aeciospores. These spores can affect *Ribes* but not pine. When leaves of the former host are affected, orange-yellow pustules appear which contain urediospores. The latter infect *Ribes*, and the urediospore cycle may be repeated during a growing season. On the lower side of *Ribes* leaves, later in the summer, brownish columns of teliospores are formed; the teliospores germinate to form sporidia. The sporidia, in turn, are borne by air currents and upon germination infect pine needles. The presence of both *Ribes* and pine in a favorable environment is essential for perpetuation and spread of the fungus.

The disease was first found in the United States in 1906, and later findings indicate that it was first introduced into New England in 1898 on nursery stock from Europe. No restriction on importation occurred between 1898 and 1912, and undoubtedly many shipments of infected stock were imported. Since *Ribes* spp. occur concomitantly with five-needled pines, the only other requirement was favorable environment. Since 1906 the disease has gradually spread across the natural range of *Pinus strobus* to western Minnesota and western North Carolina. It appears to have been introduced into British Columbia in 1910 and was found in western United States in 1921. Established first on western pines in Idaho and Montana, it has progressed in Washington, Oregon, and California.

Blister rust causes damage to old trees, but it is probably most severe on young stands which are not protected by eradication of *Ribes*. This disease stands as a classic example of a relatively unimportant pathogen endemic on the Eurasian continent, which was introduced to America during the period of unrestricted interchange of nursery stock, where it encountered susceptible hosts and favorable environment. Its spread has cost millions of dollars in losses on mature stands, and it is an ever-present threat to young trees. Its control through eradication of the alternate host is discussed later in this chapter.

Chestnut Blight. This disease was first noted on American chestnut in the New York City zoological park in 1904. The causal fungus (p. 338) was new to science, and the disease was soon recognized as a threat to the American chestnut forests, which extended from New England to the southern Appalachians of North Carolina. The pathogen invades all parts of the

tree and kills bark and cambium, causing cankers which girdle the trunk and branches. The fungus, an Ascomycete, produces pycnospores, which are water-borne and may be distributed by birds, and ascospores, which are discharged into the air and carried by air currents. The American chestnut (*Castanea dentata* Borkh.) and the European chestnut (*C. sativa* Mill.) proved to be very susceptible, while the eastern chinquapin (*C. pumila* Mill.), the western chinquapin (*Castanopsis chrysophylla* DC.), and some of the chestnuts cultivated for nuts are more or less resistant. The fungus spread by natural means in spite of interstate quarantines, and it was soon evident that the chestnut forests of the eastern United States were doomed. Small and large trees were killed rapidly until now the forest for all practical purposes has been completely eliminated. Stumps of trees with killed tops commonly send up healthy shoots, and for a time there was hope that these shoots were more resistant and might be the basis of a new forest. Abundant inoculum and favorable environment results in the destruction of new stands, however, and hopes of the renewal of stands of the American chestnut have been abandoned. The synthesis by hybridization of a suitable resistant forest tree is a future possibility.

The occurrence of chestnut blight in a city park near an important port pointed at once to the possibility of the importation of the fungus. This question was settled when Frank N. Meyer, an agricultural explorer of the Office of Plant Introduction of the U.S. Department of Agriculture, discovered the disease on native chestnuts in China in 1913. It is now generally agreed that the fungus was introduced into the United States on nursery stock from the Orient. In its native habitat the fungus is not regarded as a destructive pathogen, probably because species grown there are highly resistant. This indicates that through previous centuries susceptible individuals have been eliminated and only resistant forms survive. This is another classical example of an inconspicuous pathogen which was carried across an ocean barrier by man and introduced before any quarantine restrictions were imposed by the United States. Establishment was conditioned by a host species native to the United States which proved to be very susceptible and by the fact that the environment was at least as favorable as the habitat from which the pathogen came. Favorable environment and ready means of distribution of the pathogen precluded its restriction and eradication after establishment. Had strict international quarantine been in force, it is quite possible that its introduction would have been prevented. The loss of our chestnut forests and the prevention of their reestablishment for future generations represent values which probably exceed the monetary cost of all past and much of future quarantine in this country.

Citrus Canker. Citrus crops are not native to the United States, but they have become our most important subtropical fruits, and the crop is of immense value annually in Florida, California, and Texas. Citrus canker was first observed in Florida in 1912. The disease was new to science, and the bacterial pathogen, *Xanthomonas citri* (Hasse) Dows., was not described finally until 1915. It was probably introduced from the Orient on nursery stock between 1908 and 1911. It was recognized as a serious menace to citrus culture about 1914. It has been found on *Citrus* spp. in Alabama, Mississippi, Louisiana, and eastern Texas but not in the important citrus area of the lower Rio Grande valley and not in Arizona and California. Both the long-time quarantine in force in California since before 1890 and the relatively unfavorable environment have probably been factors in keeping it out of the valuable orchards of that state. The Federal Quarantine Act of 1912 facilitated the prevention of further importation of citrus nursery stock from abroad. The disease is now known to occur in China and India, in one or both of which countries the pathogen is probably native. It also occurs in the Philippine Islands, Hawaii, North Australia, New Zealand, South Africa, and Madagascar. State quarantines have prevented further introduction into Florida, South Texas, Arizona, and California. The pathogen has become endemic on relatively less important citrus plantings in Alabama, Mississippi, and Louisiana.

Immediate, thorough, and continuous eradicative measures have eliminated the pathogen from Florida. This is discussed further under Eradication. Such eradicative measures have made quarantine and embargo effective. The disease is not so severe on the trees of citrus as is chestnut blight on the American chestnut. It consists in scabby spots and small cankers on all above-ground parts and causes objectionable blemishes on the fruit. Grapefruit and sweet orange are the most susceptible species, but trees of these forms are not commonly killed. The climatic factors most favorable are relatively high temperature and precipitation during the period of most rapid growth of the citrus tree. These conditions prevail in Florida and in the other Gulf states, but the climatic conditions in Arizona and California tend to be less favorable (6).

Potato Black Wart. The disease was first described in Hungary in 1895. The pathogen is a fungus which attacks subterranean stems and tubers of potato and has been transmitted experimentally to tomato and some other close relatives. It has become a major disease of potatoes in most European countries. The fungus is disseminated chiefly with infected potato tubers. It persists for several years in infested soil. In England the seriousness of the disease is such that areas in which it is known to occur are quarantined, and potatoes grown within such an area must be consumed

therein. The disease was discovered in Newfoundland (then distinct from Canada) in 1909. It appeared in eastern Canada in 1918 and not again in that country until 1941, when it was found in a small garden near Halifax, Nova Scotia, in which case appropriate quarantine measures were taken promptly. Although a strict embargo of potatoes coming into this country from countries where wart was known to occur was maintained after the inauguration of the Federal Plant Quarantine Act of 1912, the pathogen became established in a few localities in certain counties of Pennsylvania, where it was found in 1918, and in West Virginia and Maryland. Fortunately, the disease occurred only in home gardens in areas which were not accustomed to export potatoes to other localities. There is good circumstantial evidence that the pathogen had been established long before 1912 and that it had been introduced by miners from central Europe who brought some of their favorite seed potatoes with them.

It had been known for some time previous to 1918 that some varieties of potatoes were highly resistant to wart. Some are sufficiently resistant to be commercially successful but are a dangerous means of spread, because resting spores may form in small inconspicuous warts.

Upon the recognition of the infected area a local quarantine was established by cooperative arrangement of Federal and state authorities. This quarantine prevented movement of potatoes out of infected localities. It also required those wishing to raise potatoes for local use to use highly resistant varieties. This quarantine is still maintained. No other infested areas have been discovered, although a large portion of American potato acreage is planted with susceptible varieties. The embargo on potatoes from countries where wart occurs has never been lifted. This case appears to be one in which local quarantine has served to restrict an important pathogen after its introduction and establishment, and strict international embargo has prevented further introduction.

Wheat Flag Smut. Flag smut is a disease of the stem, culm, and leaf of wheat. The fungus (*Urocystis tritici* Koern.) gains access to seed and straw during harvest and is distributed as a seed contaminant or on straw or other material which has become contaminated. The fungus infects seedlings from the soil, and, unlike most cereal-smut fungi, it persists for many years in infested soil without access to the host.

It was first reported in Australia in 1868. It occurs in Japan, China, India, South Africa, Egypt, Italy, and Spain. It was found in 1919 to be established in an area near St. Louis in the states of Illinois and Missouri. It has also been found in Kansas and in the Pacific Northwest. It was first thought that the importation of wheat for milling purposes from Australia in 1918 during World War I may have been the occasion of the introduction. Later evidence showing that it occurred in Missouri in the crop of

1918 rendered the above explanation unlikely. Federal Quarantine 59 excluded wheat and wheat products from India, Japan, China, Australia, South Africa, Italy, and Spain.

Dutch Elm Disease. Dutch elm disease came into prominence in western Europe about 1920. It reached severest proportions in Holland, where the most important species of elm, *Ulmus hollandica* var. *belgica* (Burgsd.) Rehd., used widely for dike holding, is very susceptible. The species and varieties of elm native to North America are also very susceptible. The organism is an Ascomycete which invades the xylem vessels, where it sporulates abundantly by yeast-like budding. The fungus is thus transported rapidly within a given vessel. All degrees of symptoms occur from slight dwarfing of leaves through wilting of leaves, yellowing, defoliation, killing of branches or of an entire tree. The disease cannot be distinguished with certainty by macroscopic symptoms, laboratory examination being essential for complete diagnosis. The fungus is disseminated by bark beetles, the chief carriers in the United States being the smaller European elm bark beetle (*Scolytus multistriatus* Marsh.) and the native elm bark beetle (*Hylurgopinus rufipes* Eich.).

Elm disease was first discovered in Ohio in 1930, when three large shade trees in Cleveland and one in Cincinnati were diagnosed as affected with the disease and were removed. In 1931 four more trees were found in Cleveland. No new cases were noted in 1932, but in 1933 an active center was found in New Jersey and New York in the vicinity of New York City. By 1938 nearly 25,000 infected trees were found in New Jersey, over 8,000 in New York, and over 600 in Connecticut. Aside from the intensive area where the disease had become endemic, a few trees were found in widely scattered cities in the northeastern United States. By 1935, 33 had been located in Cleveland, only one in Cincinnati, four in Athens, Ohio, and 88 in Indianapolis. By 1938, two were located in Baltimore; three in Brunswick, Maryland; one in Cumberland, Maryland; five in Wiley's Ford, West Virginia; and five in Norfolk, Virginia.

The widely scattered almost simultaneous outbreaks of the disease in spite of some 20 years of strict international quarantine of nursery stock was the cause of surprise and alarm. It was discovered that elm logs had been imported from Europe, unimpeded, for some years previous to the outbreak. Entry had occurred through several Atlantic and Gulf of Mexico ports. Moreover, from some of the ports they had been transported for several hundred miles on open flatcars to veneer mills. In this manner some 14 distinct terminal areas were exposed, together with some 12,000 miles of railway rights of way. Obviously, some of these logs were still harboring the blight pathogen, and the adult borers were liable to leave the logs to deposit the pathogen in growing elms. These facts explained adequately the source

of widely scattered areas in which the disease developed. More recent identification of the disease in eastern Canada indicates that it was probably introduced there by similar but distinct importations.

Federal Quarantine 70 was established to prohibit further importation of elm logs, and Quarantine 71 prohibits movement of elm trees without permit. In the New York City area there are estimated to be at least 25 million elms. Fifty per cent are small seedlings or swamp elms of no value; twenty-five per cent are of value as timber only; 6 million are shade trees. It is in the last group that the heaviest losses will be suffered. Much effort and money was expended between 1933 and 1942 in an attempt to restrict the pathogen and to eradicate it. This program was abandoned in the latter year, and direct prophylaxis was recommended as the only feasible means of control, until suitable resistant shade-tree elms are synthesized by breeding. The extent of the area in which the pathogen will become established as a serious threat to elms remains to be determined; it is dependent upon the relation of environment to the pathogen, its vectors, and the development of the disease.

This pathogen is an example of one which was introduced in spite of strict quarantine of live planting stock and illustrates how inert plant carriers may be extremely important means of introduction.

Voluntary Inspection and Certification

In general, compulsory inspection and certification before movement of planting stocks is confined in the United States to trees, shrubs, and bulbs. The increasing recognition of the importance of seeds, tubers, and transplants as carriers of pathogens has given rise to systems of inspection and certification. In general, these are to be distinguished from compulsory inspection of trees and shrubs required by state law by the fact that they are voluntary on the part of grower and distributor. In some states the rules are set up and administered by the same agency which carries out enforcement of compulsory regulation, but in others the procedures have become a part of seed-inspection organizations set up to cover certification for varietal purity, viability, freedom from weeds, etc.

One of the largest traffics in certified seed now in force has to do with potato seed tubers. Since the potato tuber may harbor many viruses as well as fungus and bacterial pathogens, reduction of these pathogens is extremely vital to successful potato culture. Long before the promulgation of certification rules and means of enforcement, it was recognized, first in the British Isles and later on the European continent and in the United States, that the more northerly areas commonly provided healthier and more productive seed tubers. The principle of control through production of seed in a "disease-proof" area is an old one and has been applied success-

fully not only to potatoes but to other vegetables. It was at first assumed that production of seed in such an area was sufficient guarantee of control. In the case of potatoes, and gradually in the case of most other vegetables and their pathogens, it was found that other measures including inspection, roguing, clean foundation stocks, etc., were necessary.

In the case of potato seed certification, a minimum number of field and postharvest inspections are required. For each inspection a maximum percentage of affected plants or tubers for each disease is allowed. Other requirements consist in good cultural practices, adequate insect control, and isolation of seed fields and bins sufficient to prevent current spread of pathogens from other fields or bins. Even these stipulations have been found inadequate because of the limitations of detecting plants affected with certain viruses, owing to the masking of symptoms when certain weather conditions prevail. This has led to additional regulations in which samples of planting stocks are subjected during the previous winter to greenhouse or southern out-of-door indexing for virus content. To provide even greater effectiveness, some states maintain a continuous supply of elite foundation stocks built up and maintained by trained plant pathologists under a rigid system of propagation of tuber units.

Most cereal and cotton seed-certification rules include disease standards, and in some states treatment of certified seeds is required. Among vegetables, tomato seed certification is most extensive.

Certification of sweet-potato roots used for producing sets and of sets ready for shipment is provided in most southern states. Certification of tomato plants is carried out extensively in the southern states, where millions of plants are produced annually for use in the middle and northern states.

Inspection of Perishable Fruits and Vegetables

In a large geographical area like that which comprises the United States many perishable food products are grown hundreds or thousands of miles away from the localities in which they are consumed. Previous to 1917 heavy losses were incurred annually in transit. In the main, these losses were due to infection of products in the field and to inadequate control of environment during transit. Under the emergency need of food conservation during World War I, a system of inspection of perishables at major loading points and at principal market terminals was set up under the Bureau of Markets, U.S. Department of Agriculture. These regulations are now administered under the Production and Marketing Administration of the same department.

Any shipper or receiver of perishable food products may secure, for a small fee, inspection of the loaded consignment at the loading point or

upon arrival at destination, or at both points. The inspection certification, which becomes a matter of public record, specifies, among other things, the health of the product including the specific designation of the diseases present and their extent. Such record is valid in any litigation which may arise over disease losses in transit between shipper and receiver or between either one and the public carrier.

The effect of this system of inspection has been to reduce immeasurably, and to maintain at a low level, disease losses in transit. As a result higher quality fresh fruits and vegetables are available throughout the year at lower cost to the consumer than might otherwise obtain. The effect of the regulations on control has been exerted (*a*) by virtually demanding shipment of only sound merchandise; (*b*) by making it worth while for the carrier to provide the best known methods of loading and refrigeration and the best transit schedules; (*c*) by protecting the receiver and indirectly the consumer from unnecessary losses and higher prices due to careless packaging and inefficient transportation.

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ERADICATION

Eradication of the pathogen is carried out in a wide variety of ways. Some of the oldest methods have to do with cultural practices such as crop rotation. In some procedures eradication cannot be separated readily from direct prophylaxis. This is true particularly with chemical treatments, such as seed treatments, soil treatments, and spraying or dusting of foliage with fungicides. For this reason eradication by chemical means is treated along with direct prophylaxis in Chap. 16. Eradication of wild or other oversummering or overwintering hosts including alternate hosts is another means of control, which like quarantine measures requires at times legal regulation and enforcement to be effective.

Crop Rotation

The rotation of crops is an old practice based quite as much upon the art as upon the science of agriculture. The scientific basis for a given rotation is often lacking. There prevails in some quarters almost a superstition that continued culture of one annual crop is detrimental to the soil. On the other hand, forest-tree crops sometimes occupy the same soil for centuries, and the tree-fruit cycle is usually a matter of decades. As a matter of fact, in some regions certain annual crops succeed as well or better in continuous production. Notable examples are onion and tobacco, while in some regions wheat and cotton are cropped very closely.

It is obvious that continuous cropping provides the opportunity for perpetuation and intensification of pathogenic organisms. The assumption that such is the case has led to a common citation of crop rotation as a remedy for plant disease. However, in current literature it is probably the most abused control measure, since it is so often recommended without consideration of whether the pathogen or pathogens concerned would be reduced effectively by such a procedure.

The first question to consider is whether the pathogen is a soil inhabitant or merely a transient soil invader (5). A **soil inhabitant** is an organism which is capable of residing in the soil for a period of several years (5 or more) without access to its host. This long tenure may be due to long-lived spores or more often to the ability of the organism to subsist as a saprophyte in competition with the soil flora and fauna. Common examples of the first group are the onion-smut organism and the crucifer-clubroot organism. Examples of the latter group are the potato-scab organism; the *Rhizoctonia* damping-off and stem-canker fungus; the various vascular fusaria and many other species of *Fusarium;* damping-off and root-rot fungi, such as species of *Pythium* and *Aphanomyces.* In most of these cases rotation is of little value in controlling the disease. Either the soil, once inhabited, remains infectious indefinitely, or the period required for elimination is too long to have any practical value.

Soil invaders, on the other hand, are as a rule not long persistent. Many of them survive only so long as the host residue persists as a substrate for their saprophytic existence. When forced to exist in soil in competition with soil inhabitants, they disappear rapidly. In this group are many parasites of annual plants. A few which may be mentioned are the bean-anthracnose organism, *Colletotrichum lindemuthianum* (Sacc. & Magn.) Briosi & Cav.; the cabbage-blackleg organism, *Phoma lingam* (Fr.) Desm.; the bean-bacterial-blight organisms, *Xanthomonas phaseoli* (E. F. Sm.) Dows. and *Pseudomonas phaseolicola* (Burk.) Dows.; and the cabbage-black-rot organism *Xanthomonas campestris* (Pam.) Dows. In regions where freezing weather prevails during the winter months, these organisms

commonly persist for 1 or 2 years. In the main, they can be eliminated as a source of inoculum by a 3- or 4-year rotation with crops which are not hosts.

In a few instances systematic research has been carried out upon the effect of rotation of a given crop with other crops upon the persistence of various pathogens in the soil. In the case of the cabbage crop it was found that in soils once heavily infested with the yellows organism, *Fusarium oxysporum* f. *conglutinans* (Wr.) Snyder & Hansen, the pathogen remained viable over a period of 12 years or more in the absence of susceptible hosts in the rotation. This organism is a soil inhabitant. The clubroot pathogen is likewise long persistent, but there is no evidence that it carries on a saprophytic existence, and it probably remains viable as resting spores. Rotations with lapses of 10 years without the presence of the host have been observed to reduce but not eradicate the pathogen. The black-rot and blackleg organisms persist in northern areas with severe winters for 2 years but seldom beyond that time, and practice indicates that a 3- or 4-year rotation of cabbage with noncruciferous crops eradicates these pathogens satisfactorily, provided noninfected seed is used.

The effect of rotation on the incidence of diseases of canning pea was studied by means of a systematic survey of their occurrence under different lengths of rotation in Wisconsin in 1942 (12). The incidence and severity of anthracnose, *Colletotrichum pisi* Pat., and bacterial blight, *Pseudomonas pisi* Sackett, decreased in proportion to the number of seasons between pea crops, although the rate of descent was modified by the factor of annual introduction of the pathogens upon seed. The incidence of *Mycosphaerella* blight, *Mycosphaerella pinodes* (Berk. & Blox) Stone, had a similar trend, but the rate of descent was influenced not only by the introduction of the pathogen on seed but also by the introduction of wind-borne ascospores as inoculum from other fields. The root-rot disease, *Aphanomyces euteiches* Drechs., also receded with lengthening of the period between pea crops, but reduction was at a much slower rate than with bacterial blight and anthracnose, probably because the pathogen is capable of persistence as a soil inhabitant either as oospores, as a very mild facultative parasite on the fibrous roots of various other plants, or as a free-living saprophyte. Which of these factors is the most important has not been ascertained. The pea crop, therefore, is one which requires fairly long rotations to be successful, because it is essential to keep the pathogens mentioned in abeyance. However, as is usually the case with rotation as a control (a factor which is commonly overlooked), the benefits from the practice may be partly or entirely offset by introduction of pathogens with seed and by failure to recognize that, when the pathogen is air-borne, it may be transmitted the following year from old fields to new fields. Pea growers are therefore advised not only to rotate the crop but to arrange the rotation so that the current-

season crop is removed as far as possible from the location of the crop of the previous season.

A study was made in western Nebraska of the relation of length and type of rotation of potatoes on irrigated land to the incidence of scab, the *Rhizoctonia* disease, and *Fusarium* wilt, *Fusarium solani* var. *eumartii* (Carp.) Snyder & Hansen (7). Here the pathogens are soil inhabitants and are not wind-borne. Of particular interest in this study is the fact that the type of rotation was as important as length of rotation, if not more important. Of the various 4- and 6-year rotations used the diseases were the least prevalent with those in which alfalfa preceded potatoes. It was pointed out that alfalfa preceding sugar beets, however, may result in more damping-off of the latter. It is important, therefore, when sugar beets and potatoes are in rotation with alfalfa, that the latter immediately precede potatoes rather than sugar beets.

In southeastern United States tobacco is an important cash crop which is commonly grown in short rotations. Four important soil inhabitants which affect the crop are the incitants of southern bacterial wilt, *Pseudomonas solanacearum* E. F. Sm.; stem rot, *Pellicularia rolfsii* (Curzi.) West; *Fusarium* wilt, *Fusarium oxysporum* f. *nicotianae* (J. Johnson) Snyder & Hansen; and root knot. Rotation experiments conducted by Clayton *et al.* (2) have yielded some interesting results. Tobacco following bare fallow had the least amount of root knot, but the yield was usually lower than that of more severely diseased tobacco following corn or tobacco. Although nematode-immune crops, such as peanuts, crotalaria, and oats, tended to reduce the nematode population in the soil, they were no more effective in control of root knot than bare fallow and did not necessarily result in better yields. Although vetch was very susceptible, tobacco following vetch grown as a winter crop was practically free of root knot. The results with bacterial wilt were different. Bare fallow had no effect, while the culture of corn for a year greatly reduced the disease. Although the causal organism may infect many cultivated plants and weeds, there was no correlation between population of susceptible weeds and the incidence of wilt in subsequent tobacco. The stem-rot organism has a wide host range, and there was found to be no relation between the type of rotation and occurrence of the disease. *Fusarium* wilt was observed to increase in rotations in which sweet potatoes preceded tobacco.

Another example of the effect of previous crops and crop management on the importance of a disease is furnished by the work of Garrett and Mann (6) on the take-all disease of cereals (*Ophiobolus graminis* Sacc.) in England. Previous experiments had shown that the chief requirement for rapid dying out of the pathogen from infected stubble residues seemed to be a shortage of available nitrogen. In a comparison of six autumn treatments of stubble

two effects of nitrogen were distinguished: (*a*) an immediate effect in assisting survival of the fungus in infected root and stubble residues and (*b*) a deferred effect in promoting disease escape of the following crop. Ploughing in of straw in the autumn was found to increase take-all, presumably because the locking up of available nitrogen by the decomposing straw withheld it from the following crop and this factor outweighed the beneficial immediate effect, *i.e.*, starvation of the fungus by depriving the latter of nitrogen. An autumn growth of trefoil (*Medicago lupulina* L.) on the stubble seemed to be entirely beneficial to the following crop; active growth of the legume appeared to assist in starving out the fungus, and in the spring the release of nitrogen by decomposition of the trefoil benefited the barley crop which followed immediately.

Sanitation

Sanitary measures with respect to plant pathogens are feasible when the life history of the organism concerned permits a rational and effective procedure. Removal of plants as they become diseased is often recommended without due consideration of whether such a measure is practical and effective. Removal of cabbage plants affected with clubroot or yellows, for instance, would be of no value, because more of the persistent pathogen in each case would be left in infected roots in the soil than would be removed with the readily accessible portion of the diseased plant. Clean culture in the apple orchard reduces the amount of inoculum of the scab organism in dead leaves by covering them with soil, but practice shows that it is only a part of essential control. There are, however, many instances where sanitation is worth the cost and effort.

The potato-late-blight organism overwinters chiefly in infected tubers. When such tubers are dumped from warehouses onto refuse piles in the spring months, they become a dangerous source of primary inoculum. Many tubers sprout; the fungus attacks such sprouts, sporulates, and in favorable weather produces sporangia, which, being readily disseminated by air currents, become the source of initial infection in the growing crop. Such refuse piles commonly attract maggot flies, which lay eggs in the rotting tubers. The larvae become contaminated with soft-rot (blackleg) bacteria, and the next brood of flies, being contaminated, is prone to lay eggs in the field near emerging potato plants. The larvae of the next brood, still contaminated, serve as vectors of the bacteria as they feed upon the potato seed piece. Blackleg epidemics may thus be initiated. If tubers in refuse piles are infected with aphid-borne viruses, such as mild mosaic and leaf roll, they commonly serve as a source of virus inoculum. Aphids often colonize in early spring on potato sprouts and in that way become viruliferous. As that particular source of food wanes, the winged

females migrate, often to the growing plants of the current-season crop. In these ways a carelessly placed refuse pile may become the source of several disease epidemics, and sanitation is therefore important in the proper disposal of this and many other types of plant refuse which harbor plant pathogens.

Some pathogens are more readily distributed by handling practices than are others; some depend largely on such practices for their dissemination. One of the important pathogens so transmitted is the potato-ring-rot organism. The bacterium concerned is spread from place to place in seed tubers but from tuber to tuber in a given locality by handling practices. Organisms emerge in slimy masses from infected tubers, and in this form they contaminate crates, sacks, grading machinery, cutting knives, planting equipment, and the hands of workmen. Thus one or a few infected tubers in a lot of several hundred bushels may provide inoculum to contaminate and infect many more tubers in the same lot and further to contaminate other lots, originally entirely free, which may be passed through the same handling equipment. Sanitation in this case consists in decontamination of bins, containers, equipment, and workers between seed lots, in addition to procedures designed to prevent the introduction of contaminated lots into the warehouse. Sanitation in fruit and vegetable packing houses is important. Decontamination by fungicides is discussed in a later chapter.

Some viruses are very readily spread by mechanical means. Two important ones are the common-tobacco-mosaic virus and the latent potato virus. Individually either of these viruses causes a relatively mild disease effect upon greenhouse tomatoes; in combination they cause the very severe disease known as streak. Both viruses are long persistent in vitro, and thus workers' hands, becoming contaminated, serve as a continuous source of inoculum. Tobacco-mosaic virus is commonly spread through the tomato crop in greenhouses by the pruning and tying operations. If hands also, by chance, become contaminated with potato latent virus, a streak epidemic will follow the pruning operation. The tobacco-mosaic virus may be picked up from cigarettes or chewing tobacco. It is also readily spread in the same way by workmen weeding tobacco beds. In these instances important sanitary measures consist in assuring that workmen keep hands decontaminated by frequent washing in soapy water and avoid handling of smoking or chewing tobacco and potato tubers when working in tomato or tobacco crops.

There are many more cases of sanitation which might be cited, but those mentioned above will show the type in which this method of control is important. The student of plant pathology should recognize that recommendation of a sanitary measure is meaningless unless it is based on scien-

tific facts. The same procedure may be extremely effective and therefore valuable for one pathogen but entirely useless for another, depending upon how it may affect the cycle of the organism or virus concerned.

Elimination of Overwintering Hosts

When the host plant is an annual, and when the pathogen or virus does not overwinter (or oversummer) on debris or in the soil, wild perennial hosts often become the locus of overwintering. Many of the downy-mildew fungi overwinter as mycelium in the crowns of perennial weeds. The cucumber-mosaic virus is carried over winter in milkweed, pokeweed, and many other plants. The potato-yellow-dwarf virus and the potato-rugose-mosaic virus (veinbanding virus) are carried over in *Physalis* spp. The yellow-bean-mosaic virus is carried over in sweet clover. The cabbage-mosaic viruses are carried over in shepherd's-purse and peppergrass. Many other viruses might be cited. When such plants can be eradicated economically, the cycle of the pathogen or virus may be broken and the disease controlled.

In the main, eradication of overwintering hosts is not feasible, usually because the cost is too great. In some cases the high value of the crop may justify the expenditure. Control of cucumber mosaic by eradication of wild hosts was studied in Wisconsin and Illinois (1916-1924) by Doolittle and Walker (3). They were of the opinion that control of mosaic was practical in most sections if a combination of eradication of weed hosts, rotation, and isolation was practiced. Wellman (13) was of the opinion that eradication of vines of *Commelina nudiflora* near celery fields in northern Florida was a feasible means of destroying the source of the southern-celery-mosaic virus. Here, where the acreage is so concentrated and the crop so valuable, the eradication procedure appears to be feasible. Recently this program has been enhanced by the use of chemical weed killers, particularly 2,4-D.

Avoidance of the overwintering source of inoculum is sometimes much more practical than eradication. In Wisconsin, eradication of wild hosts to control cucumber mosaic has never come into practice. However, the commercial industry has avoided the disease to a large extent by moving the acreage northward and dispersing it, so as to secure more isolation and to avoid building up of wild host reservoirs. In Iowa and in Oregon the yellow-dwarf disease of onion became destructive where onion sets were grown near onion seed plants which, in turn, had grown from bulbs infected the year before. Aphids carry the virus from seed plants to young seedlings. The sets grown from seed were used the following season to produce a crop of bulbs. Thus seed, sets, and bulbs were grown in close proximity, and bulbs served as the source of early-season inoculum. This disease was controlled in Iowa by a regulation which provided for the growing of sets in isolated areas free from seed onions. In Oregon, where commercial seed

and bulb crops were important, each crop was restricted to an area, and the two areas were separated sufficiently to prevent contamination of the bulb crop from bulbs overwintered for seed production.

In the Puget Sound section, where cabbage seed is grown, the biennial nature of the crop provides a continuous growth of cabbage as seedlings or as seed plants in a relatively small geographical area. The cabbage-mosaic viruses are thus carried through the winter on plants destined to produce seed, and aphids transmit them the following summer to seedlings growing to produce heads for the next year's seed plants. The mosaic viruses thus permitted to build up continuously became very destructive. Pound (10) showed by a series of experiments that the disease could be reduced to a minimum if the cycle were broken by growing seedbeds in isolated areas removed from the seed crops and protecting the seedlings from infection until after transplanting. A similar procedure was successful in the Venice district of Los Angeles County, California, where year-round culture of celery led to heavy losses from celery mosaic. In 1934, a celery-free period from July 31 to Jan. 31 was invoked voluntarily by growers and was authorized the next year by the state legislature. The elimination of the host during the time of year when the crop was least valuable reduced the disease effectively and restored normal yields in the remaining months as a result of disease control (9).

Eradication of Plant Parts; Tree Surgery

The excision of cankers in woody plants to remove the pathogen is sometimes found to be a practical measure, in that it reduces the source of primary inoculum and precludes further advance of established cankers. Lesions caused by fire blight on pear and apple trees are sometimes removed during winter months and the exposed wood treated with a fungicide to prevent reinfection. Heart rot of valuable shade trees can sometimes be excised to prevent further advance of the pathogen, and life of the tree may be extended. In all such measures the value of the tree is an important consideration, and complete excision of affected tissue is essential to success. Ark (1) described eradication of crown gall on almond trees by the application of elgetol (sodium dinitrocresol) to the gall and contiguous healthy bark.

Eradication of Host and Pathogen

Peach Yellows and Peach Rosette. These virus diseases of peach, especially yellows, have plagued peach culture in the eastern United States for over a century. With peach yellows, fruits have abnormally red spots, and leaves roll upward along the margins and downward along the midrib. The yellow foliage and sickly decline of the tree result in death in 4 or 5

years. Yellows occurs throughout the eastern United States but not in the Pacific Coast states nor outside of North America. Rosette occurs chiefly in southeastern states and not west of the Rocky Mountains. It appears early in the spring. Large leaves drop, and twig growth is stunted to produce conspicuous rosettes of leaves. Most affected trees die after a single summer. Smith demonstrated about 1890 that both diseases were due to virus infection, but he could transmit the viruses only by budding and grafting. The serious threat to peach culture occasioned by the rapid spread of these diseases resulted in the inauguration of control by eradication (11). As soon as either disease was detected in nursery or orchard, it was recommended that the diseased tree be removed and destroyed. Throughout the past 50 years this has been practiced on a voluntary basis in orchards and as a part of inspection regulation in nurseries. It still is the only feasible control and appears to have successfully checked the two diseases. In 1933, Kunkel brought out proof that peach yellows may be transmitted by the leaf hopper *Macropsis trimaculata* Fitch., which is known to occur from Ontario and Maine as far south as Pennsylvania and Iowa and as far west as Colorado.

Citrus Canker. The introduction of the citrus-canker organism into southeastern states and the quarantine measures which followed have already been described under Exclusion. The imminent danger to the citrus groves of Florida was the occasion for a thorough eradication program carried out by Federal and state authorities beginning about 1915. All nurseries and orchards were inspected by crews which followed strict sanitary precautions against spreading the bacterial pathogen. When an orchard or nursery was found to contain an infected tree, the entire planting was eradicated by burning the trees with kerosene torches. Canker was found on 515 properties scattered through 26 counties. About 3 million trees were destroyed. No infected trees have been reported since 1927, although inspection of plantings has been continued. This is an outstanding example of a large-scale eradication program which was entirely successful. A Federal and state inspection and eradication program in eastern Texas was initiated more recently. In 1945, the Chief of the Bureau of Entomology and Plant Quarantine reported that no trace of the disease could be found in Texas at that date, and Federal inspection was withdrawn.

Phony Peach Disease. This is a virus disease of peach which became important in the Fort Valley, Georgia, area about 1915. It has a stunting effect upon the tree, and fruits are reduced in size. It is a disease of the root system, and the virus can be transferred by grafting diseased roots upon healthy peach roots. In 1948 erratic occurrence of the virus in the tops was shown, and a leaf hopper vector was announced. At first restricted to the Fort Valley area, the disease was later found in several

southern states. An eradication program was begun in 1929. This program consisted in (*a*) inspection of nurseries in Gulf coast states; (*b*) inspection and eradication of trees in commercial orchards. In 1935, out of over 6 million trees inspected from South Carolina to Texas, 103,000 affected trees were located. About 100,000 trees were eradicated in Georgia alone in the years 1935 and 1936. In 1945, the Bureau of Entomology and Plant Quarantine reported that since 1936 the virus had apparently been eradicated from six entire states and reduced by 98 per cent in eight others. Recent demonstration that the virus may occur in certain wild species of *Prunus* without inciting symptoms and the discovery of an insect vector complicate the problem of permanent eradication.

Eradication of Alternate Hosts

Barberry Eradication (4). In some of the destructive diseases in which long-cycle heteroecious rust fungi are concerned, the eradication of the less valuable host is a possible means of control. Long before De Bary established the heteroecious nature of the black-stem-rust fungus, growers of wheat in Europe and America recognized some importance in the relation of the barberry to this disease on grain. The first law requiring the destruction of barberry is said to have been passed at Rouen, France, in 1660 at the instigation of farmers in that area. At approximately the same time the disease appeared in New England, where colonists had probably introduced it on barberry, which they brought to America with them and planted widely as a hedge plant. Beginning in 1726, there were enacted in the colonies of Connecticut, Massachusetts, and Rhode Island several local laws which recognized the prevailing opinion that the barberry was in some way related to wheat rust and which provided various means for eradication of the bush in view of the alarming increase in the destructiveness of the disease. These laws apparently were not rigidly enforced and, being subject to considerable opposition, gradually lapsed or were repealed. Many local eradication laws were passed in various European countries during the nineteenth century. By 1923 there were 25 local laws in various subdivisions of Germany. A law of 1869 in Denmark was superseded by a more stringent one in 1904. Norway passed a law in 1916; Hungary, in 1920. In England, voluntary eradication has been successful.

After the lapse of the colonial laws no action was taken in America until the twentieth century. Severe rust epidemics in 1916, coinciding with expanding demands for American wheat in World War I, resulted in a series of state and Federal acts which established the barberry-eradication program, which is still under way, in the northern half of the United States. In the period from 1917 to 1923 South Dakota, North Dakota, Iowa, Montana, Minnesota, Nebraska, Michigan, Wisconsin, Wyoming, Oregon, and Wash-

ington passed barberry-eradication laws, all of which are still in effect. Beginning in 1918, Congress passed a Federal appropriation to assist in the eradication and has continued to support it annually since then. The Federal program was administered by the Bureau of Plant Industry until 1934, when the work was transferred to the Bureau of Entomology and Plant Quarantine. In 1918, a barberry-eradication area was established through cooperative agreement of the Federal Bureau and the enforcement agencies of the wheat area comprising the states of Montana, Wyoming, Colorado, North Dakota, South Dakota, Nebraska, Minnesota, Iowa, Wisconsin, Michigan, Illinois, Indiana, and Ohio. The Federal Horticultural Board in 1919 established Quarantine 38, which prohibited shipment of 31 rust-susceptible species of *Berberis* and *Mahonia* into the area; it did not restrict interstate movement within the area, this being handled by individual state regulations until 1931, when the Federal quarantine was extended to interstate movement. In 1937, the eradication area was enlarged to include Missouri, Pennsylvania, Virginia, and West Virginia. Canada began comparable legislation in 1917, which eventually applied to the contiguous provinces of Manitoba, Saskatchewan, and Ontario.

The eradication program has been a mammoth and a costly one. To be fully effective, a systematic survey during summer months of every property in township, village, city, and county was necessary over nearly a million square miles of territory. The common barberry had become, since the time of its introduction into America by the colonists, a widely planted dooryard shrub. Had it remained a dooryard and hedge plant, systematic eradication would have been relatively simple, though great in scope. However, the fruits being common food of birds, seeds were disseminated widely, and the plant found many favorable environments in northern states. Wild barberries in many areas have become the most difficult and costly to eradicate, particularly when rough terrain is involved. Recurrence of shoots from surviving roots of removed plants required rechecking after a few years had elapsed. By 1942 over 300 million bushes had been destroyed, and about 60 per cent of the entire area was practically free from barberry. About 40 per cent had been covered once and still required rechecking. It was estimated at that time by the Bureau of Entomology and Plant Quarantine that the damage to cereal crops in the eradication area had been reduced by about 60 per cent.

In this, as in most obligatory eradication programs, the air-borne nature of the pathogen requires that the program be practically complete in order to be effective at all. In the case of black stem rust of cereals in the northern states, success of eradication is conditioned upon the nonsurvival of urediospores during the winter. In the winter-wheat areas of Kansas, Oklahoma, and Texas, there is little to be gained because of urediospore sur-

vival. The eradication of the barberry has two main benefits: (*a*) it eliminates the early spring primary inoculum, and (*b*) it prevents the synthesis of new or nonprevalent physiologic races of the fungus through hybridization of monocaryonts prior to production of aeciospores on the barberry. The control of stem rust by barberry eradication is limited by the fact that epidemics may arise from urediospores carried in by air currents from southern locations where such spores survive the winter climate. By and large, the benefits of the campaign have justified the cost of the program to date and of continued surveillance in the future.

White-pine Blister Rust (4, 8). As already stated in the discussion of quarantine, the white-pine-blister-rust fungus was introduced repeatedly from Europe on white-pine seedlings from 1898 to 1912. In 1909 one shipment from Germany was distributed to 226 localities. By 1911 diseased stock had been distributed in New Hampshire, Vermont, Massachusetts, Connecticut, Pennsylvania, Indiana, and Ohio. Early attempts to eradicate these pines were abandoned, because it was obvious that the pathogen was already established on native pines and *Ribes*. A series of Federal and state regulations followed which were designed to restrict the movement of pines and of currants and gooseberries. Another phase which soon began to assume greater significance was the systematic eradication of *Ribes*. In some 20 states there had been designated by 1942 certain blister-control areas selected upon the basis of the value of the white pine and upon the willingness of property holders to cooperate in an eradication program. In these control areas and in a surrounding zone at least 1,000 ft. in width, *Ribes* was to be eradicated to prevent production of sporidia from teliospores which might infect pines. By 1942, 28 million acres of land had been included in blister-rust-control areas, of which about two-thirds had been covered by an initial eradication survey and about one-fourth had been rechecked at least once.

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CHAPTER 16

DISEASE CONTROL THROUGH PROTECTION

While exclusion and eradication have importance in keeping pathogens from becoming established, all crops are subject to many unfavorable factors in the environment and to many pathogens which are already present and which initiate diseases on susceptible hosts as soon as environment is favorable. As already indicated, the two major lines of disease control are (*a*) direct prophylactic measures designed to prevent infection and (*b*) immunization of the host by breeding or by other means. Many prophylactic measures involve the application of fungicides to seeds, foliage, or other plant parts. There are prophylactic measures of lesser scope which will be considered first.

REGULATION OF ENVIRONMENT

The importance of environment in disease development has already been brought out in previous chapters. The control of disease by regulation of environment at first thought appears to be farfetched. On the contrary, it is used so often and so subtly that its importance is generally overlooked. To be sure, the grower of crops has little control over the weather, but it is well for the student of plant pathology to take time to consider how much the art of agriculture is influenced by the relation of climate to disease.

Selection of Growing Areas. It has already been mentioned under Exclusion (p. 610) that most of the certified seed potatoes in the United States are now grown in the northern tier of states. The chief reason for this is that the environment is generally more favorable to the reduction of current-season spread of viruses by insects. Most of the seeds of cabbage, turnip, and rutabaga grown in the United States are now produced in Pacific coast areas, although formerly they were produced in middle western and eastern localities. The major reason for the shift in growing region is the fact that the two most important seed-borne pathogens, the blackleg organism and the black-rot organism, do not become established readily in the areas concerned because of the very low rainfall during the period when the young plants are growing in crowded seedbeds, while in middle western and eastern areas, it is almost impossible to produce seed without heavy infection at times. For the same reason bean seed growing has been shifted to irrigated Rocky Mountain and Pacific coast areas. Here the factor of dissemination by spattering rain is greatly reduced. This is sufficient almost to eliminate the bean-anthracnose organism and to reduce the bacterial-blight organisms. Pea seed was once grown chiefly in the northeastern quarter of the United States, but production has in the

past 30 years shifted to western areas, along with that of bean seed and for the same reason. Thus we find in these and in many other cases that might be cited that it is more feasible to control certain diseases by shifting the crop to an environment unfavorable to the diseases concerned than to regulate environment where the culture of the crop is already established. This is particularly applicable to production of seed, where the total acreage of the crop is relatively small in proportion to the crop for which the seed is used, and where the healthy condition of the seed is highly important to economical production of that crop.

The shifting of growing regions is not confined to seed crops. A study of the adjustment of agricultural production in the United States and in the world shows that many growing areas have always been more or less temporary. Many factors are involved in these transitions, but disease epidemiology is not an unimportant one of the factors. Cantaloupe production in coastal regions is often unprofitable, because environment is favorable to downy mildew. Fire blight is so prevalent and destructive on desirable commercial varieties of pear that many areas in the Middle West and East which might otherwise produce them have long ago given up commercial culture, while areas in California and Oregon which are somewhat less favorable to blight produce most of the crop at present. In the discussion of control of cucumber mosaic by eradication of weed hosts, it was pointed out that in Wisconsin there was a shift of commercial acreage to the northern half of the state, where the crop could be dispersed more widely and greater isolation and longer rotation provided. In the pod-pea-growing area in the upper Snake valley of Idaho, where the high altitude increases the frost hazard, the acreage is concentrated on tableland near the mouths of mountain canyons, where the natural drainage of warmer air during frosty nights protects the growing crop from injury. Air drainage is particularly important on muck tracts and in low-lying creek bottoms, particularly when the cultivated tracts are surrounded by dense stands of trees. Onion mildew and cucumber scab (*Cladosporium cucumerinum* Ell. & Arth.) are particularly favored by poor air drainage, presumably because of the greater prevalence and duration of mists and dew. On this account avoidance of certain fields or tracts is sometimes practiced for these crops.

Storage and Transit Environment. The regulation of storage and transit conditions has received much attention during the past three decades. Much of the fruit and vegetable produce now grown is shipped long distances and often stored for considerable periods before it is consumed. In general, fungus and bacterial pathogens are restricted in their activity as the temperature is reduced. Low temperatures, therefore, are

most commonly provided for disease control. Many factors enter into the picture, however, which determine the most satisfactory temperature for the fruit or vegetable concerned. While temperatures near 0°C. are desirable to restrict pathogens, the host plant sometimes reacts unfavorably to such temperatures. Irish potatoes sweeten and sometimes discolor, and sweet potatoes develop internal necrosis, at temperatures between 0 and 5°. Too high a humidity promotes sprouting of onions; too low a humidity causes shriveling of carrots. Gaseous emanations may accumulate to the point where they cause scald of apples. It has therefore become necessary to determine the conditions most satisfactory for transit and storage of all important perishable products. When these facts are known, proper regulation of storage environment is conducive to disease control as well as to maintenance of marketable and culinary quality of the products.

Greenhouse Environment. Glasshouse growing of flowers and vegetables is based on the production of a product of high value under extremely intensive cultivation. The environment commonly provides conditions of humidity and temperature quite distinct from those of outdoor culture. Some diseases prevalent outdoors are absent or nearly so under glass, while other diseases minor in importance outdoors can be very destructive under glass. An example may be found in the tomato, which is grown in the northern states as a summer field crop and indoors as a winter crop. Early blight, leaf blotch (*Septoria lycopersici* Speg.), and late blight are common foliage blights outdoors but are practically absent under glass. Leaf mold (*Cladosporium fulvum* Cke.), on the other hand, is absent in the case of most northern crops in the field but is universally present and often destructive in greenhouses. The regular daily periods of high humidity, which increase steadily during the autumn months, bring about an environment required for infection and sporulation by *C. fulvum*. The only successful control of the disease is by thorough and timely ventilation to restrict favorable humidity. In the fall months fires are started in tomato greenhouses as early as August to provide enough heat to stimulate aeration and reduction of humidity. When skillfully handled, this measure usually provides practical control and is practiced widely in tomato houses.

CULTURAL PRACTICES

Many cultural practices have come into use which tend to reduce the progress of diseases. Avoidance of cultivation of row crops while dew is present on the foliage reduces the spread of water-borne spores and bacteria. It has long been a sound procedure with beans in the control of spread of anthracnose and bacterial-blight organisms. Shallow planting of potatoes in soil where the *Rhizoctonia* disease is prevalent serves to reduce the stem-

canker phase. This is accomplished by covering the seed lightly in a furrow of regular depth and throwing soil to the plants after growth is well started when the plants are less susceptible to infection.

Time of Sowing and Planting. Timing of sowing or planting may sometimes be helpful by avoiding periods particularly favorable for a pathogen. Early sowing of fall wheat in the Palouse area of Washington and Idaho has been recommended because the higher temperature of the soil is unfavorable to germination of chlamydospores of the wheat-bunt fungus and consequent infection of the seedlings during their period of susceptibility (p. 385).

Regulation of Soil Moisture. Some diseases are particularly enhanced by excessive soil moisture or by irregular water supply. *Aphanomyces* root rot of pea is incited by a soil water mold which requires rather high soil moisture for infection and disease development. The selection of well-drained fields and avoidance of poorly drained spots are highly essential factors in keeping this disease in abeyance in pea-growing areas of Wisconsin and other northern states (139). The control of blackheart of celery has been shown by Foster and Weber (40) to be dependent upon a regular water supply in which sudden extremes of high and low soil moisture are avoided. In northern Florida, subirrigation of celery lands provides a means of controlling this disease. Blossom-end rot of tomato is also associated with irregular water supply, and a simple corrective measure in home gardens is the use of a grass or straw mulch during the latter part of the growing season to reduce fluctuation in soil moisture.

Defoliation of cranberries in Wisconsin and Massachusetts soon after the removal of winter flood (used to protect vines from freezing injury) is attributed to a gradual depletion of oxygen under the ice during the previous winter and consequent smothering of the plants. It has been determined that this damage can be controlled by either of two methods. The flood water is drained off as soon as 8 to 10 in. of ice have formed, following which the ice sinks into the vines, still protecting them and reducing the danger of suboxidation. The second method is to freeze the vines into the ice by adding water gradually and allowing it to freeze until the ice is thick enough to protect without damage (125).

Some fungi that persist indefinitely in the soil can be reduced by flooding the area concerned, where irrigation or runoff water is available. In the southern states the "drop" fungus is a troublesome soil inhabitant, since it attacks many truck crops and rotation has little value. Brooks (18) has shown that in Florida, where artesian-well irrigation is often available, flooding for 6 to 8 weeks during the summer destroys about 90 per cent of the sclerotia. In Central America the banana-wilt organism, *Fusarium oxysporum* f. *cubense* (E. F. Sm.) Snyder & Hansen, infests the soil and

causes the abandonment of large areas for culture of this crop. Wardlaw (140) reports that experiments have been conducted wherein abandoned areas were flooded for periods of from 6 to 18 months. When the areas were then replanted, little sign of disease had appeared after 5 years' production, and it appears that the treatment would permit a still longer period without further treatment.

In Egypt, flag smut (*Urocystis tritici* Koern.) is troublesome on wheat when soils become infested with the pathogen. There are two prevailing methods of sowing the crop on irrigated soils. The "herati" method consists in broadcasting seed on moist land and plowing it in. The "afir" method involves broadcasting the seed on dry soil, harrowing it, and then irrigating. It has been shown that the percentage of infected plants is less when the latter system is used. It is considered that the wet soil discourages infection, while shallow planting shortens the amount of susceptible host tissue exposed to the pathogen (39, 63).

Adjustment of Soil Reaction. In a few diseases the adjustment of soil reaction is a means of control. The potato-scab organism is widely distributed in potato soils, where, like many other saprophytic members of the same genus, it persists as a soil inhabitant. Highly acid soils are unfavorable to this organism, and fortunately potatoes produce satisfactorily in soils with a pH as low as 5.0. Scab is very appreciably reduced when the pH is below 5.2. Many potato soils are naturally in this range of hydrogen-ion concentration. This is particularly true along the Atlantic seaboard from Maine to Florida. By the use of acid-forming fertilizers and acid-tolerant cover crops and by avoiding the use of lime in quantities which decrease the acidity to pH 5.2 or above, scab has been effectively controlled. Where soils of limestone origin are used, the reaction is often favorable to scab. It is usually possible to raise the acidity of such soils to a range inhibitive to scab and thus bring the disease under control. However, this procedure has had little commercial application so far in the United States because of cost and because of interference in crop-rotation schedules (28). Sweet-potato soil rot, *Streptomyces ipomoea* (Person & Martin) Waksman & Henrici, is severe in Louisiana in soils with reactions of pH 5.6 to 5.8 or above. Application of sulfur to the soil to change the reaction to pH 5 or lower practically eliminates losses (100).

Extreme acidulation of sandy soil in northern Florida has been found to provide control of another pathogen, the southern-bacterial-wilt organism. This affects many hosts in subtropical and tropical regions, particularly potato, pepper, eggplant, and tobacco. The first three of these are commonly grown in rotation as winter crops, and since the pathogen is a soil inhabitant, losses are sometimes heavy. Eddins (37) has worked out a procedure wherein the soil is made extremely acid (pH 3) by application

of sulfur and is allowed to remain in this condition during the summer months, when the wilt organism, being intolerant to this extreme acidity, is greatly reduced in amount. In the autumn, the pH is again raised by addition of lime to about pH 5 before the potato crop and to pH 5.5 before the eggplant or tomato crops are planted. By this means a considerable measure of control is attained.

The addition of lime to soil to control clubroot of crucifers has been practiced for two or three centuries (p. 160). Here the pathogen is known to be retarded in germination and infection if the soil solution is kept at pH 7 or above. Liming of the soil is a satisfactory control measure on some soils. In muck soils the buffer capacity is such that it is impracticable to maintain a pH sufficiently high to control. In other cases on mineral soils, alternating high and low soil moistures sometimes permit infection in spite of heavy liming (74). Where potatoes are grown in rotation with crucifers, liming is impracticable because it provides favorable conditions for potato scab.

Regulation of Fertility Level and Balance. Acute disease development sometimes arises from a disproportionate amount of one or more major mineral elements. Potash deficiency is quite common. Potash hunger in many crops is corrected by fertilizer control. Predisposition to disease sometimes follows potash hunger. One of the outstanding cases is that of cotton wilt, *Fusarium oxysporum* f. *vasinfectum* (Atk.) Snyder & Hansen, which tends to be more severe in plants growing on potash-deficient soil. There are a few other situations where disease may be reduced by direct control of soil fertility. Application of nitrogen to apple orchards is a common practice to encourage a proper carbohydrate-nitrogen balance in the tree. Nitrogen tends to promote succulence in growing twigs, and such tissue becomes more susceptible to fire blight than less succulent growth. Caution in the use of nitrogenous fertilizers is thus a very essential part of apple-orchard management. On some soils in the southeastern states nitrogen fertilizers have a depressing effect on bacterial wilt of tobacco (123). In California nitrogenous fertilizers tend to reduce the damage to sugar beet following infection by *Pellicularia rolfsii* (Curzi) West (76).

Correction of Deficiencies in Trace Elements. The correction of mineral deficiencies is an important control measure. On some soils where boron deficiency is likely to prevail, borax is commonly added before planting. This is true for canning beets in Wisconsin and New York and for alfalfa, apple, and many other crops in various regions. In Ontario, boron is so readily fixed during the growing season that rutabagas are sprayed with borax solutions or suspensions to prevent brown heart. Some muck soils must be corrected for copper deficiency and some for manganese deficiency before susceptible crops are sown. Copper deficiency is corrected by

adding copper sulfate to fertilizers. If a copper fungicide has been applied to the previous crop, there probably is enough copper available for the current crop. Beans are commonly sprayed with manganese sulfate solution on everglade muck soils to correct manganese chlorosis. Corrective applications can also be applied with fertilizer. In various sections of the Atlantic seaboard magnesium deficiency is corrected by the application of dolomitic lime. In the case of potatoes in that region the use of such lime in the preparation of Bordeaux mixture serves to correct magnesium deficiency. Zinc deficiency in citrus and pecan is corrected by the addition of zinc sulfate to the soil, or it may be applied by incorporation in a spray mixture. Iron-deficiency chlorosis is a disease which affects vine- and tree-fruit crops, particularly where the soil is alkaline and the iron is fixed in a form unavailable to the plant. The fixation of manganese, boron, zinc, and molybdenum also occurs in alkaline soils. Where it is practical to change the reaction to neutral or slightly acid by application of sulfur or of acid-forming fertilizers, these elements tend to become available, and deficiencies are often corrected. However, acute disease development is more satisfactorily prevented by annual applications of the required salts in soluble form as a soil amendment or as a very dilute spray.

HANDLING PRACTICES

The importance of sanitation in the handling of propagative materials, such as potato tubers, and in the packaging of fruits and vegetables has been discussed in a previous section. Other important measures of control of diseases which affect harvested crops will be considered next.

Contamination of the crop by storage pathogens can sometimes be reduced greatly if the life cycle of the pathogen is considered. An important example is the late-blight rot of potato. The disease develops first on the foliage, and sporangia, mycelium, or zoospores may migrate through the upper layers of soil to the tubers. This latter process, however, is often checked or entirely prevented by unfavorable weather for the fungus at the critical time. If potatoes are harvested at a time when the foliage still harbors the fruiting fungus, the opportunity for tuber contamination and subsequent infection is greatly enhanced. Moreover, if tubers are still not quite mature, the skins are wounded more readily in handling, and infection by the blight fungus is favored still more. In areas where frost is imminent at harvesttime or before, a few nights of temperature just below freezing may kill tops and fungus sufficiently to remove the inoculum. Vine-killing chemicals and flame throwers are commonly used to kill late-maturing foliage to accomplish the same end. The handling practice which serves most to control late-blight tuber rot, therefore, is the delay in digging of the crop, if weather permits, until above-ground inoculum is reduced to a

minimum and until the skin of the tubers below ground has matured to the maximum stage.

The avoidance of bruising of potatoes during harvest is important, because wounds are an important avenue of infection by tuber-rotting fusaria. The potato-tuber tissue is normally very reactive at this time, and cork-cambium developments set in rapidly around wounds. The cell walls of the cork-cambium tissue become suberized, and penetration of wound parasites is inhibited. It has been shown that this cork-cambium activity proceeds more rapidly at about room temperature than at storage temperature. Prestorage curing of potatoes for about 2 weeks at about 21°C. is recommended before they are placed in storage. It has likewise been shown that prestorage curing of sweet potatoes is necessary to stimulate rapid corking over of wounds to prevent infection by several of the pathogens which cause storage rots. The crop is cured at about 32°C. in high relative humidity for about 10 days before it is placed in permanent storage at 10 to 15°. This is a particularly important practice with sweet potatoes because storage at temperatures near 0° to reduce fungus action is not feasible, since a nonparasitic breakdown occurs at temperatures below 10°.

Onion neck rot is an important storage decay in which infection takes place at harvesttime. Penetration is facilitated by exposure of the succulent tissue when the top is removed at harvest. The percentage of infected bulbs will be reduced if the tops are allowed to mature thoroughly at the neck of the bulbs before harvest operations are started. The "sealing" of the bulb tissue by this natural process is the best means of control. If moist weather persists at this critical period, the fungi concerned may infect before complete desiccation of the neck tissue occurs. Incipient infection can still be checked effectively by rapid drying through rapid artificial circulation of warm dry air. As a rule the latter control measure is practiced only where the value of the crop is very high. In the main, handling procedures at harvest, properly applied, are adequate to control this disease.

Precooling of fruits and vegetables before shipment is sometimes practiced in order to check promptly incipient infection which might otherwise progress during the lag period required to reduce the temperature of a carload of produce by icing the bunkers.

CONTROL OF INSECT VECTORS

When the infectious agency is usually introduced or disseminated by an insect vector, the control of the latter becomes a major part of the prophylaxis. This sometimes consists in sanitary measures, sometimes in the application of insecticides.

Now that the Dutch-elm-disease organism is established in certain parts

of the United States, the prevention of further spread within such areas is dependent upon the success with which the insect vector is suppressed. The overwintering brood of beetles emerges from diseased elms or from dead or cut trees and brush in May and June. All diseased trees and infested wood should therefore be burned before May to dispose of the vectors.

The cucurbit-wilt organism overwinters in adult cucumber beetles, which infest young plants as they emerge from the soil. Early and thorough application of insecticides is essential to prevent the establishment of this disease. It is not usually possible to prevent it entirely by this means and by a subsequent spray or dust program which controls insect damage. Clayton (22) has shown for Long Island conditions that an appreciable reduction in wilt results from a thorough insecticidal program.

While it might be expected, since insects are the chief disseminating agents of most viruses, that insecticides would be the chief means of control of this group of diseases, it has seldom worked out so in practice. In the main, the successful control of the insect in so far as insect damage is concerned does not control the disease incited by the virus which the insect carries. The only satisfactory explanation of this fact is that, generally speaking, even with good insect control, enough insects escape to bring about a virus epidemic. Up to the present at least, most insecticides are not efficient enough to control virus diseases. Perhaps improvement in insecticides will bring about a better solution of this problem. There are a few exceptions which might be cited. In greenhouses control of aphids by fumigation may be so complete as to prevent spread of cucumber mosaic. Pound and Chapman (103) used DDT spray on carrots to control aster yellows through reduction of the hopper vector. They were able to reduce the percentage of diseased plants sufficiently to increase the yield, by 20 per cent or more in some cases.

HISTORY OF THE DEVELOPMENT OF FUNGICIDES

The use of fungicides as protectants developed slowly and empirically long before the germ theory of disease was well established. It was not until the last half of the nineteenth century that, concurrent with expansion of the modern concepts of parasitism, definite progress in their development occurred. Sulfur had been known as a therapeutic agent since the time of Homer, at least 1,000 years before the Christian era. Democritus in 470 B.C. recommended sprinkling pure amaca of olives on plants to control blight. Practically no successful procedures were in general use before 1600.

Remnant (109), in 1637, mentioned seed treatment for wheat. In 1705, Homberg (58) recommended mercuric chloride as a wood preservative.

In 1716 a patent was issued in South Carolina for a wood-preserving process by means of "oyle or spirit of tar" for preservation of the "bottom of vessels from the river worm and also the plank from rotting" (58). In 1733, Tull (135) described a salt-brine treatment for wheat seed which had been in use since the previous century. In 1755, Aucante (7) mentioned arsenic and mercuric chloride as seed-treatment materials to control bunt, and in the same year Tillet (131) described reduction of wheat bunt when seed was treated with liquid containing salt and lime and with saltpeter solution. In 1761, Schulthess (116) used copper sulfate solution on wheat seed, as did Tessier (128) in 1783. De Boissieu and Bordenave, independently, recommended copper sulfate as a wood preservative in 1767 (58). Thus there had been at least a century of empirical testing of various materials for seed treatment and wood preservation when Prévost (104), in 1807, showed by laboratory studies that the spores and sporidia of the wheat-bunt fungus were killed or inhibited by copper sulfate solution.

Wood preservatives and methods of treatment were improved in the first half of the nineteenth century. Wade suggested zinc chloride as a wood preservative in 1815, and a patent covering its use for the purpose was issued in 1838. The early process consisted in the immersion of wood in solution in open tanks. A pressure process was devised in 1847. A patent on the use of mercuric chloride as a wood preservative was issued in 1832, and one for the use of copper sulfate in 1837. The coal-tar-creosote process was first patented in 1836, and in 1838 a patent was granted on the injection of creosote under pressure (58). After about 1850 application of these measures to railroad timbers increased in importance.

One of the first modern records of a fungicide being used to spray or dust foliage was the claim of Robertson (113), in England (1824), that sulfur was specific for powdery mildew of peach. Kenrick (70), in the United States, boiled lime and sulfur together and proposed its use for grape powdery mildew in 1833. In England in 1834, Knight (55) recommended the sprinkling of peaches with sulfur and lime to control leaf curl. Teschemacher (127), a chemist in New England, studied the late-blight rot of potato and suggested in 1845 the application of common salt and possibly other salts to the soil to control the disease. Cheever (2) in New York suggested in 1845 that, since copper sulfate was effective against grain rust (probably referring to seed treatment for wheat bunt), it should be incorporated in the soil to check potato-late-blight rot. Morren (95), in Belgium, recommended in 1845 a mixture of lime, common salt, and copper sulfate as a soil treatment for the control of potato tuber rot associated with late blight. In 1846, it was noted in Wales that late blight was not present on potatoes near a copper smelter, while those farther away were seriously affected (3). About 1850 a method was described in the United States by

Hays (4) for reducing late-blight rot in potatoes in storage. Crude sulfur was inflamed in a cast-iron or earthen vessel, and the fumes were introduced into the air spaces surrounding the potatoes in the bins.

Powdery mildew of grape was introduced into Europe from America about 1845, and the disease rapidly became destructive in vineyards. Duchartre (88) in 1848 recommended the use of sulfur dust as a control, and the remedy was so effective that it came into widespread use for this and other powdery-mildew diseases. Grison, head gardener of the forcing houses in the kitchen garden at the palace at Versailles, France, boiled lime and sulfur together and applied the diluted solution as a spray for vine powdery mildew with good success. It was described in 1852 (52) and became known as "Eau Grison" but apparently did not supersede the more satisfactory sulfur-dust treatment. Radclyffe, authority on rose culture in England, reasoned that, since copper sulfate was effective in the treatment of wheat seed for bunt, it should be satisfactory as a spray for powdery mildew of rose. He reported a successful trial in 1861 (105) but later found the solution so toxic to rose foliage that he withdrew the recommendation. Dreisch (35) is commonly cited as the first to report the addition of lime to copper sulfate to reduce its injury to wheat seed (1873). However, it is certain that this practice began much earlier, since Bollman (15), in an article entitled "The Wheat Plant" in the first annual report of the U. S. Department of Agriculture (1862), cited as a common remedy for wheat bunt in America "soaking the seed wheat in washes of different kinds, among which is that of dissolved bluestone, having considerable strength, during one night, and then mixing quick lime with the still wetted wheat. Another is to use salt instead of bluestone, soaking the same time and followed by the same application of quicklime."

Discovery of Bordeaux Mixture

The downy-mildew disease of grape appeared in France in 1878, where it was observed by Millardet in a nursery on seedlings from America. It is generally agreed that the fungus was introduced from the United States on nursery stock, for at that time there was much activity in controlling the *Phylloxera* gall louse, to which certain stocks from America had been shown to be highly resistant. Downy mildew was not controlled by sulfur dust, and the extreme susceptibility of French varieties of grape resulted in the rapid development of severe epidemics in many intensive grape-growing areas. In 1882, Millardet, professor of botany at the university of Bordeaux, France, noticed that downy mildew was held in check when vineyardists in the intensive grape-growing district in the vicinity applied a liquid containing lime and copper sulfate to vines near the roadways to discourage thieves from stealing fruit. Following up this lead, he an-

nounced Bordeaux mixture in 1885 (94) and it rapidly gained wide acclaim in the grape-growing regions of Europe. Thus, although copper sulfate had been used for 100 years or more, and although it had been observed that when it was mixed with lime its toxicity to wheat seed was reduced, the combination of the lime and the sulfate had not been used on foliage. This proved to be an epochal discovery, since Bordeaux mixture was adopted rapidly in both Europe and America not only for the control of downy mildew of grape, but also for late blight for potato, apple scab, black rot of grape, and numerous other diseases.

Limitations of Bordeaux Mixture

As Bordeaux mixture came into extensive use in the 15 years immediately following its discovery, agriculturalists and investigators became increasingly aware of certain of its limitations. Chief of these was its phytotoxicity on certain species of plants and under certain climatic conditions. **Phytotoxicity** of a fungicide is the property of causing one or another type of injury to the plant to which it is applied. These injuries, in the case of Bordeaux mixture, are now known to vary in form and degree and are influenced by environmental conditions. Some of the effects are blemishes on the fruit, as in apple; foliage injury, as in peach; retarded growth, as in cucumber; excessive transpiration resulting in drought injury, as in ginseng and some vegetables; blossom drop, resulting in delayed production, as in tomato. Before the end of the nineteenth century search had begun for other spray materials which might be equal to Bordeaux mixture in fungicidal value and lower in phytotoxic value.

Rise of Sulfur Fungicides

The next significant development was concerned with sulfur compounds. As already stated, sulfur dust was the first fungicide to be used extensively on foliage. By 1860 its application was an established procedure, and it was eminently successful against powdery mildews. Outside this group of diseases its adoption was negligible, and it was of no value against downy mildews and late blight of potato. Boiled lime and sulfur, as we have pointed out, received only incidental attention. In 1905 Parrott, Beach, and Sirrine (98) reported from New York State that "lime-sulfur wash," made by boiling lime and sulfur together, reduced apple scab somewhat, but they used it as an early spray followed by two applications of Bordeaux mixture. Cordley of Oregon is generally credited with revival of this material as a fungicide, and it rapidly became known as "lime-sulfur solution." In 1908 he reported marked success in apple-scab control following its use (25). He emphasized in his first report that he secured better control of scab than with Bordeaux mixture, while fruit blemishes caused by the

latter were avoided. It should be pointed out that Cordley was working in an irrigated area where rainfall was low during the growing season. From this time on, the use of lime-sulfur expanded rapidly, and it shared with Bordeaux mixture the first place among fungicides used as foliage sprays. There followed development of other types of sulfur and copper sprays and dusts which will be described later.

Revival of Dusts

G. C. Johnson of Kansas City, Missouri, introduced a powder form of Bordeaux mixture in 1900. In 1902, Tubeuf (134) used copper carbonate dust on wheat seed for the control of bunt. Dusting, however, remained in the background for another two decades. In 1917, Darnell-Smith (29), in Australia, revived the copper carbonate dust treatment for wheat seed. It was rapidly adopted in the United States under the impetus of increased need for wheat during World War I. Sanders and Kelsall introduced copper-lime dust (a mixture of finely divided calcium hydrate and monohydrated copper sulfate) as a substitute for Bordeaux mixture in 1918. Beginning about this time, sulfur dust received more attention, and its fungicidal properties were improved by greatly reducing the size of particles through a variety of methods. While copper and sulfur dusts have come to share a place with sprays in the field of protective control, they do not equal liquid sprays in the volume used.

Development of Organic Fungicides

Up to 1913 the materials used as fungicides were inorganic chemicals, with the exception of formaldehyde. In 1913, Riehm (110) in Germany, following the success of Ehrlich in developing organic arsenic for syphilis in man, announced the value of chlorophenol of mercury as a treatment of wheat seed for the control of bunt. This was the beginning of a broadening of fungicidal materials beyond the range of copper and sulfur compounds. For the first 25 years, the organic-fungicide field was confined largely to seed disinfectants. The successful compounds were largely organic mercury compounds, chiefly applied as dusts. In about 1936 emphasis was shifted to seed protectants (53, 55, 56). Again inorganic compounds held sway for a time because of their lower cost. Red copper oxide was foremost among these, with zinc oxide being as effective and somewhat cheaper for some seeds, *e.g.*, spinach. The opening of World War II placed a premium on copper, zinc, and mercury, and this competitive demand stimulated the search for new, purely organic fungicides. Several outstanding materials were found which took the lead as seed-treatment dusts. Chief of these were chloranil, or tetrachloroquinone, marketed under the trade name, Spergon, and tetramethylthiuram disulfide, which

is the active component of Tersan, used as a spray on turf grasses, and of the seed-treatment dust marketed as Arasan in the United States and as Sulsol in England. Numerous new organic compounds have shown promise as foliage sprays. Chief in prominence among these are the carbamates discussed in more detail later.

FOLIAGE SPRAYS AND DUSTS

Fungicides as sprays and dusts, and less commonly as fumigants, are applied to the foliage of plants usually as **protectants.** In other words, they are designed to be present at the infection court in advance of the pathogen in order to prevent infection. While acting in this role, they may also have a direct effect upon organisms which have already invaded the host. In this case they act as **eradicants** or as **suppressors of sporulation.** As a rule, protective fungicides are of relatively little value as eradicants. They are sometimes modified, however, to act in this role, or special materials may be used for this purpose.

Eradicants

There has been relatively little attention given to fungicides as suppressors of sporulation. There are indications given by some investigators that paradichlorobenzene acts in the control of downy mildew in tobacco seedbeds as a sporulation inhibitor. The addition of sodium arsenite to Bordeaux mixture applied to apple leaves in the late autumn was used experimentally by Keitt and Palmiter (64) to kill the apple-scab pathogen in infected leaves or so modify it that the ascigerous stage was prevented from forming. Various dormant sprays have been used to reduce the amount of inoculum in overwintering lesions, particularly on trees. Ground spraying of orchards to reduce sporulating inoculum by preventing the maturation of ascospores and the discharge of those which have formed at the time of application is another eradicant method (65). Treatment of powdery mildews with sulfur dust is, in part at least, a procedure of killing the fungus after infection has occurred.

Protectants

Protectant fungicides, to be successful, must have certain characteristics. Since they are applied to foliage to protect the latter over a period of time, they are usually exposed to rain and dew. This means that the material must be nearly insoluble in water but still soluble enough to function as a fungicide. Tenacity or adhesiveness is another important feature. The fungicide must adhere well in order to have a lasting effect. It must cover the surface well, in order to protect the entire exposed area. These important qualities have been the basis of rise or fall of many a substance which

has been brought into the field. They have led to much research not only upon the fungicidal property of materials but upon their physical properties and upon compatibility with modifiers which may be added to the mixtures to improve their tenacity and coverage. These latter substances are commonly referred to as **spreaders** and **stickers.** Few fungicides are free from phytotoxicity, and some host plants are more susceptible than others to injury by a given material.

There are now a large number of materials used as fungicides which, as they have become perfected, have often been found to have special value under certain circumstances. Thus, at the present time, the trend is away from emphasis upon an over-all single fungicide. Rather, in recognition of the range of host and pathogen sensitivity, specialization in materials is under way. Most of the fungicides applied to foliage as sprays, dusts, or fumigants can still be placed in one of three major groups on the basis of the dominant metal or nonmetal in their composition. These groups are (*a*) the inorganic copper group; (*b*) the inorganic sulfur group; and (*c*) the organic group, in which various heavy metals may or may not be attached to organic radicals.

The Inorganic Copper Group

Bordeaux Mixture (60, 88, 89). The chemistry of Bordeaux mixture is complex, and even after much research it is still not well understood. Millardet and Gayon thought there was a complete chemical reaction as follows:

$$CuSO_4 + Ca(OH)_2 = Cu(OH)_2 + CaSO_4$$

They observed, however, that the acidity was corrected before all the lime was used. Pickering and others showed that about 75 per cent of the lime equivalent required to complete the above reaction was needed to attain neutrality and precipitate all the copper. This led to the conclusion that an intermediate compound, possibly $4CuO \cdot SO_3 \cdot 3H_2O$, was formed. With the excess of lime there is a further slow reduction in alkalinity, and some investigators hold that other basic compounds are formed between $4CuO \cdot SO_3$ and the lime, giving possibly $5CuO \cdot SO_3$ and $10CuO \cdot SO_3$. Others maintain that the precipitate is a cupric hydroxide copper sulfate complex, which undergoes changes after deposition on the foliage (88).

Bordeaux mixture is a suspension which is fairly tenacious, nearly insoluble, and spreads fairly well. The fungicidal action is due to the formation of soluble copper, which is toxic to spores or sporelings in drops of atmospheric water. The factors which bring about solubility of toxic ions are still not fully understood. One possible factor is atmospheric carbon dioxide and ammonia dissolved in the meteoric water. Another factor is

secretion by the fungus, either before or after the spore germinates, of materials which promote solubility of copper-containing materials. A third factor may involve secretions from the host plant. All three may enter in, and their relative importance may vary.

Bordeaux mixture, aside from its fungicidal effects, may be beneficial or detrimental to the host. After more than 60 years of use its effects upon the host plant are still being explored. As we have noted above, certain phytotoxic effects were observed by early workers. When such effects are minor in relation to fungicidal benefits, the former are usually not evaluated and often are not even recognized. When fruit blemishes, such as the "russeting" of apples, occur in large amounts, however, orchardists are forced to substitute a less injurious fungicide. Definite benefits have been observed on grape, potato, and other plants when disease was absent or of minor importance. Greener foliage is sometimes the outstanding response to Bordeaux mixture under such circumstances. The stimulative effect of copper ions upon host metabolism has been held by some to be responsible. Increased transpiration rate has been shown by many investigators. However, the beneficial effect of Bordeaux mixture, above its fungicidal value, and its insect-repellent quality, which operates in some situations, are still conjectural.

Manufacture of Bordeaux Mixture. Early experience in the making of Bordeaux mixture led to the practice of slaking burnt lime (CaO) in water and diluting the suspension into the major portion of the water in the final mixture. The copper sulfate is dissolved in water in as concentrated a solution as possible and mixed with the lime suspension. This procedure yields a more finely divided precipitate than that obtained when lime and copper sulfate are mixed, both in concentrated solution or suspension, and then diluted. Solution of copper sulfate in half the water of the ultimate mixture and suspension of the lime in the other half before mixing is a quite satisfactory procedure and is often more convenient.

Finely divided grades of granulated copper sulfate, often referred to as "snow" and "instant," and finely divided calcium hydroxide (or mixture of calcium hydroxide and magnesium hydroxide, when dolomitic limestone is the source of material), commonly referred to as "limate," have now largely replaced bluestone and lime in the manufacture of Bordeaux mixture. Both can be added in proper amounts to the spray tank, provided with an agitator, and an "instantaneous" mixture attained. Thus the "mussiness" of homemade Bordeaux has been largely overcome.

The formulae of Bordeaux mixture vary. The original mixture contained 15 per cent copper sulfate ($CuSO_4 \cdot 5H_2O$) and 8 per cent lime (CaO). The formulae 4–4–50, 5–5–50, etc., refer to 4 lb. of copper sulfate, 4 lb.

of burnt lime, and 50 gal. of water, etc. The present usage is to base formulae on 100 gal. of water, *e.g.*, 10–10–100, 10–6–100, etc. With the use of monohydrated sulfate and calcium hydroxide, corrections to copper sulfate and lime equivalents must be made. Thus for 10–10–100, 10 lb. of granulated copper sulfate, 15 lb. of limate, and 100 gal. of water are required. Sometimes a weaker Bordeaux mixture is recommended for more sensitive foliage. In some cases the proportion of lime is reduced, where the excess $Ca(OH)_2$ is known to be the basis of phytotoxicity.

Proprietary pastes and powders designed to overcome the inconveniences of making Bordeaux mixture from copper sulfate and lime have appeared on the market in large numbers in Europe and in America. They are prepared for admixture with water for immediate use. None has been shown to be superior to homemade Bordeaux mixture. As a rule they are more costly. The large operator, therefore, usually makes up his own Bordeaux mixture. The small gardener may prefer the ready-made mix at a slightly higher cost.

Burgundy Mixture. Soon after Bordeaux mixture was developed, Masson (90) (1887) substituted sodium carbonate for lime and produced a spray for grape mildew, which was called Burgundy mixture. It has also been used under the name soda Bordeaux. Pickering found that, unlike Bordeaux mixture, the complete precipitation of copper and complete neutralization of acid ions occurred simultaneously. The blue precipitate at neutralization contains basic copper carbonate ($10CuO \cdot 4CO_2$ or $5CuO \cdot 2CO_2$) and sodium sulfate. The carbon dioxide evolved combines with sodium carbonate to form bicarbonate. In general, this mixture is about as effective in fungicidal value as Bordeaux mixture. It never came into widespread use, however, because rather than being less phytotoxic than Bordeaux mixture, it involved more risk of spray injury in use.

Other Early Copper Sprays. Attempts to improve upon Bordeaux mixture were numerous in the period between 1885 and 1930. Among the materials which came into use but which have by now been largely discarded were various combinations of copper carbonate and ammonium hydroxide and ammonium carbonate. These mixtures appeared as Eau Céleste, Modified Eau Céleste, and Cupram. Cheshunt Compound, which belongs to this group, was recommended in England (1921) as a soil spray to prevent damping-off of tomato. Basic copper acetates were also used to some extent.

Copper-Lime Dusts. As already indicated, the era of dusts to replace sprays began about the time of the introduction of copper-lime dust by Sanders and Kelsall in 1918. Renewed emphasis on sulfur dusts took place shortly before that date. The relative merits of spraying and

dusting have been the subject of many experiments since. Copper-lime dust is a mixture of finely divided monohydrated copper sulfate in various proportions with finely divided calcium or calcium-magnesium hydrate ("limate"). In the presence of moisture this forms a Bordeaux mixture precipitate on the foliage. To be most effective, it must be applied on foliage when meteoric water, preferably dew, is present. Otherwise, its adhesiveness is not great. As in Bordeaux mixture, the proportion of copper and lime may be varied. Common formulae are 20–80, 25–75, the first figure referring to the pounds of monohydrated copper sulfate, the second to the pounds of calcium hydroxide per 100 lb. of mixed dust. Spreaders and insecticides, such as arsenates, may be added also; *e.g.*, 20–20–60 copper-calcium-arsenate-lime or 25–25–50 copper-talc-lime mixtures. Copper-lime dust has been replaced largely by insoluble copper dusts which can be applied with effectiveness on dry as well as on wet foliage.

Insoluble Coppers. In 1932 Horsfall (53) reported the value of cuprous oxide (CuO) as a seed protectant, and it soon was found to have some value as a foliage spray. There followed rapidly a series of copper compounds, which as a class are commonly referred to as "insoluble coppers." The purported advantage of this group of compounds was that they did not require the addition of lime, which was being shown at about this time to be not entirely inert and somewhat phytotoxic, and that they did hold the copper in a more "fixed" form than did Bordeaux mixture. These materials vary in the percentage of copper but are ordinarily evaluated in relation to one another on the basis of the percentage of metallic copper. As a class they are not quite so tenacious as Bordeaux mixture, and they are generally somewhat less fungicidal. But since they also are less phytotoxic, they have been found to be more useful than Bordeaux mixture in many instances, especially on sensitive foliage where lower fungicidal value is offset by lower phytotoxic injury. Most of these compounds appear on the market under patents or under copyrighted names.

Cuprous oxide produced by an electrolytic process is sold as Yellow Cuprocide. A similar compound is used in England and Canada under the trade name Perenox. Basic copper sulfate ($CuSO_4.4Cu(OH)_2.H_2O$) is made from copper sulfate exposed to scrap copper in the presence of a catalyst. It is marketed under the trade names Basi-cop, Tribasic Copper Sulfate, Tennessee 26, and others. The processes of manufacture may differ somewhat. Copper oxychloride [approximately $2\text{–}5Cu(OH)_2.CuCl_2(CuO)_x$] is another material which has appeared as Copper A and Cupro-K. C-O-C-S is copper oxychloride-sulfate with the approximate formula $5Cu(OH)_2.Cu(Cl_2.SO_4)$. Some of the insolubles are applied as sprays, others are dusts; some may be used in either form (42, 83).

The Inorganic Sulfur Group

Sulfur Dusts. Sulfur dust came into use as a fungicide, especially against powdery mildews, in the first half of the nineteenth century. Present-day sulfur dusts have been improved by grinding to smaller particle size. The tendency to formation of aggregates has been overcome by addition of small percentages of inert materials such as kaolin or bentonite-sulfur. Sulfur may be active at a distance, presumably by volatilization. Contact between sulfur and fungus, however, is ordinarily necessary, and fineness of particle is important both in adherence and in toxicity. Sulfur may also be phytotoxic, causing burning, stunting, or defoliation under some circumstances. Muskmelon, for instance, is quite sensitive. Sulfur dust is still used most widely against powdery-mildew diseases. It is effective against cereal rusts but is not used widely for that purpose, primarily for economic reasons. It has been applied extensively and with success against bean rust.

Lime-Sulfur. When lime and sulfur were boiled in the early part of the nineteenth century, the fungicidal value of the resultant material was not fully appreciated. The mixture was used as a sheep-dip and as an insecticide intermittently until the phytotoxicity of Bordeaux mixture forced its revival in the early part of the twentieth century. Calcium thiosulfate and calcium polysulfides are the chief constituents. A greater part of the polysulfide is converted to free sulfur after lime-sulfur is sprayed on the foliage. This process is thought by some to be an important part of the fungicidal action of lime-sulfur.

Burning of foliage and fruits may occur in hot weather following lime-sulfur spray (36). Leaf and fruit drop may also be noted. Thus while lime-sulfur has become a very useful spray, it is not without limitations. It must be avoided for some species of plants. It has become most useful on apple but is used little if at all on vegetable crops. An exceptional case was its use at one time as an insecticide against psyllids on potatoes in Colorado, Utah, and Wyoming, but more effective insecticides have now taken precedence.

Self-boiled Lime-Sulfur. The phytotoxicity of lime-sulfur on stone fruits in the eastern United States led to the development and recommendation of self-boiled lime-sulfur by Scott (117) in 1908. This was made by using 8 lb. of sulfur flour and 8 lb. of burnt lime with 50 gal. of water. A small amount of water was added to the lime, and when slaking was well started, the sulfur was added, and the ingredients were mixed vigorously. Care had to be taken to avoid amber-colored streaks in the mixture; these contained phytotoxic polysulfides. After boiling had ceased, the remainder of the water was added. This material was less phytotoxic

than lime-sulfur largely because in it the reaction between lime and sulfur was less complete. It is no longer used, having been replaced largely by wettable sulfurs.

Dry Lime-Sulfur. This is dehydrated lime-sulfur with granulated sugar added as a stabilizer. It is said to be "safer" than original lime-sulfur because the oxidation of the polysulfide is carried out in part in the drying process, while the sugar forms a film around the sulfide particles and slows down further oxidation. This material is little used at the present time.

Wettable Sulfurs. These materials are prepared either in paste or powder form. They have come into extensive use because they cause less foliage injury than lime-sulfur. In this group are the flotation sulfurs, which are made from hydrogen sulfide gas which occurs as a waste product in the gas-coke industry. The end product is very finely divided elemental sulfur. Grinrod wettable sulfur is made by a process of emulsification and atomization of molten sulfur. The sulfur in the final preparation is in the form of very small particles. Micronized sulfur is elemental sulfur ground to fineness comparable with that of flotation sulfur. Kolofog contains about 33 per cent sulfur and 66 per cent bentonite; these are fused in the molten stage and later reduced to a powder form. Kolospray is a blend of about 85 per cent finely divided sulfur and 15 per cent bentonite (48, 83).

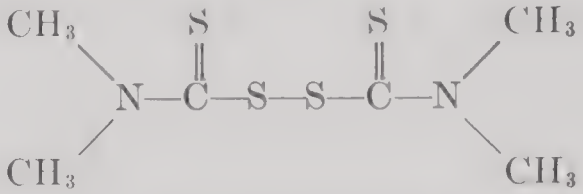
Other Sulfur Sprays. Sulfides other than lime-sulfur have been introduced without, however, coming into very extensive use. Liver of sulfur is obtained by fusion of alkali carbonates with sulfur. As in lime-sulfur, fungicidal activity is due to polysulfides. It appears to be more phytotoxic than lime-sulfur. Ammonium polysulfide was recommended for use against powdery mildew of hops and gooseberry in England. Calcium monosulfide was tried in the United States and in England but proved to be inferior to other sulfur sprays.

The Organic Group

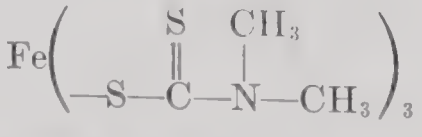
The materials in this group are placed here more as a matter of convenience than because of any natural affinities. The materials discussed under the copper and sulfur groups were all inorganic compounds. These materials have held sway throughout the nineteenth century. The only break from heavy metals and sulfur to the organic compounds in the last century was the case of formaldehyde. This is discussed under seed and under soil treatment. It is not used as a foliage fungicide. The use of organic mercury compounds began in 1913 for seed treatment. It was not until near the beginning of World War II that organic foliage sprays came

into the picture. Perhaps the discovery and patenting of dithiocarbamates in 1934 was the starting point, as nearly as we can define it. It really followed closely upon the surge of insoluble copper compounds about 1930. The great impetus came with the priorities on copper and mercury during World War II. Sulfur was never scarce during that period (112).

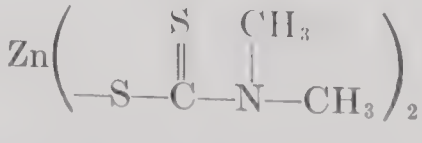
Carbamates. Investigations of dithiocarbamic acid derivatives were begun by the du Pont Company in 1931. In 1934 a patent was secured by Tisdale and Williams of that company on the use of this group of materials as insecticides and fungicides. Tetramethylthiuram disulfide, now known as thiram, has the following formula:



It is used as an activator in the production of synthetic rubber under the trade name Tuads. It was offered in the United States under the trade name Tersan as a spray treatment of turf grasses and in England as a foliage spray for tulips under the trade name Tulisan. It was also found to be valuable as a seed protectant and will be discussed in this connection later. This compound was not satisfactory as a general foliage spray, but in combination with certain heavy metals it offered much promise. One of the first compounds to be tested widely was ferric dimethyldithiocarbamate, now known as ferbam, which is marketed under the name Fermate. It has been shown to be effective against a large number of fungus diseases affecting foliage, some of which have previously been difficult to control. It appears to have high tenacity, and low phytotoxicity. The formula for ferbam is:



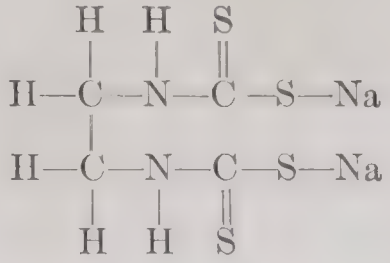
Another compound, known as ziram, is zinc dimethyldithiocarbamate with the following formula:



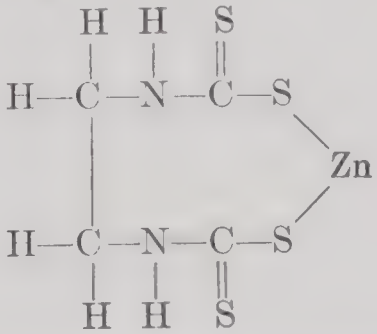
It was introduced under the trade name Zerlate.

In 1943, Dimond *et al.* (32) reported on the fungicidal value of disodium

ethylene bisdithiocarbamate, now known as nabam. It has the following formula:



It was first marketed under the trade name Dithane. The protective value of this material was later shown by Heuberger and Manns (51) to be markedly improved by the addition of zinc sulfate and lime to the mixture. This material is effective against many diseases of fruits and vegetables. The manufacturers for a time recommended the addition of zinc sulfate and lime to nabam when it was diluted to use as a spray. The zinc salt, zinc ethylenebisdithiocarbamate, known as zineb, is now used more commonly. It has the following structure:



It is marketed under the trade names Parzate and Dithane Z-78.

Quaternary-ammonium Derivatives. This group of materials has also been explored as to their fungicidal value. It was noted by Howard (57) that compounds containing nitrogen in which only three valences were attached to radicals or ions were usually of low fungicidal potency. Quaternary-ammonium derivatives, in which the nitrogen nucleus exerts five of its valences, have greater fungicidal value. When ammonium hydroxide is placed in water, it dissociates into a NH_4 cation (positive) and an OH anion (negative). Each of the four hydrogen ions is replaceable, while the OH ion can be replaced by an acid radical. Four groups of substituted compounds have been tested as fungicides. Examples of the respective groups are (*a*) Zephirin, a complex of benzyllauryldimethylammonium chloride; (*b*) Puratized N5D and N5E, phenyl mercuri triethanolammonium lactate; (*c*) Isothan Q15, laurylisoquinolinium bromide; (*d*) cationic nicotinium salts, which are combinations of six- and five-membered heterocyclic nitrogen ring compounds. A large number of compounds within each group is possible. This group of materials is

used extensively in the field of textile preservation. As foliage fungicides they are beginning to find a place in practice.

Other Organic Sprays. A wettable form of Spergon (choranil, or tetrachloroquinone) has been used successfully for the control of downy mildew of cabbage in Florida. The du Pont Semesan Company manufacture under the name Special Semesan a product containing 24 per cent hydroxymercurichlorophenol and 3.9 per cent hydroxymercuricresol for control of brown patch and dollar spot of turfs. Sodium dinitro-ortho-cresolate (Elgetol and Krenite) is used as a ground spray in orchards to eradicate or suppress sporulation of pathogenic fungi overwintering on leaves and other host refuse on the orchard floor (65). Phygon (2,3-dichloro-1,4-naphthoquinone), glyoxalidine derivatives (130, 141), and polymeric organic polysulfides (126) show promise. Sodium pentachlorophenate is used as a dormant spray on apricots in California to reduce brown-rot blossom blight. It may be expected that further research will bring forth other organic materials which will have general or special values as foliage sprays.

Fungicide Safeners

A safener is defined as a chemical that reduces the phytotoxicity of another chemical. Since many of the most effective fungicides are phytotoxic to some species of plants and under some circumstances, the value of safeners has long been recognized. The early experience with copper sulfate both as a seed treatment and as a foliage fungicide, led to attempts to "safen" it. Lime was added for this purpose when copper sulfate was used for treatment of wheat seed. The significance of the discovery of Bordeaux mixture was that it provided an effective safener for copper sulfate as a foliage spray. Lime is used universally with arsenicals to prevent the formation of or to neutralize arsenic acid, which is phytotoxic to sensitive foliage. Lime alone has some disadvantages as a safener. Zinc sulfate in combination with lime is sometimes more effective.

Glyceride oils are also good safeners for copper sprays. Supposedly the oil provides a film around each particle and thereby reduces the phytotoxic value of the particle, but it reduces the fungicidal value as well.

Spreaders

Spreaders are materials added to the spray mixture to improve the contact between fungicide and the sprayed surface. Martin (88) points out that for practical purposes we are concerned not only with the theoretical spreading property of the spray but the wetting and penetrating properties as well. Spreaders may be classified as water-soluble and water-insoluble. In the latter group are mineral oils, glyceride oils, and terpene

oils. They are limited to mixtures in which toxic constituents are soluble in oil. Water-soluble spreaders are characterized by their property of reducing the surface tension of water, a phenomenon which is now currently explained on the basis that the rather large molecule of the spreader orients itself at the surface of the liquid in such a way that the chief part of the molecule, which is water-insoluble, is at the surface while the water-soluble portion is attracted to the water of the liquid.

Soap has long been known as a spreader. Robertson (113) in 1821 recommended that sulfur be mixed with soap suds before being applied to peaches to control mildew. Soaps are alkali salts of carboxylic-acid derivatives of hydrocarbons. The longer the chain, the better the spreader. Potassium soaps dissolved in soft water are the most satisfactory. The disadvantage of soaps is that in calcium-containing fungicides or insecticides the insoluble calcium salts of the soap are formed. There has, therefore, been a great deal of research directed toward the development of soap substitutes. Many of these are now on the market. Casein and its derivatives are widely used for spray materials to which soaps cannot be safely added. Casein is a protein of high molecular weight which, being insoluble in water, is made dispersible by mixing with lime or other alkaline materials. Sulfated alcohols, sapamines, resinates, petroleum sulfonic acids, and other types of materials have been placed on the market as spreaders.

Stickers

The importance of adhesiveness of sprays and dusts when applied to foliage has already been pointed out. The term sticker is applied to a material added to a spray or dust which improves its adherence to the plant surface. Spreaders are stickers, in that they facilitate a more uniform film of spray material, from which, when the water evaporates, the residue is more adhesive than if the spray is applied in large droplets. Flour, starch, gum arabic, and dextrins are commonly used as stickers. Bentonite clays added to dusts improve the sticking quality. Oils and oil emulsions are also used. When Bordeaux mixture and lead arsenate are emulsified with cottonseed oil, the adhesive quality is greatly improved. Hydrocarbon oils have also been employed as stickers.

In general, we may expect to find that the more soluble the fungicide, the more liable it is to leach, and conversely, as tenacity is improved by the addition of stickers, the fungicidal value is reduced.

Spraying and Dusting Equipment

The first method of applying liquid fungicides and insecticides to plants consisted in dipping a whisk of straw or similar material in a container of

the material and scattering it upon the foliage. After 1880 there was a rapid improvement in spraying devices. Hand-driven syringes and pumps of various sorts were employed. The knapsack sprayer and the barrel-pump sprayer came into use before 1890. Many of the early spraying machines in use before 1896 are described by Lodeman (80). Improvements available by 1910 are described by Goodwin (46). By now there are many types of sprayers available, varying from the small hand-pump sprayer to large power-driven ones adapted to row spraying and to orchard spraying. For complete descriptions of sprayers and dusters consult McClintock and Fisher (83).

The evolution of the spray nozzle is an important phase of the development of spraying equipment. The earliest nozzles delivered unbroken streams of liquid. The modern nozzles consist of various devices to break up the liquid so that it is deposited upon foliage in finely divided droplets. Many early nozzles are described by Lodeman (80). The first "cyclone" nozzle was devised by Barnard in the United States about 1880. The liquid was forced to whirl in an eddy chamber before it passed through the outlet orifice. This was modified, to overcome a great tendency to clogging, by Vermorel, a manufacturer of spraying equipment in France. The modified form was introduced about 1887 and became widely adopted and commonly known as the Vermorel nozzle.

As the proper timing of spray applications became recognized as extremely important to successful control, the need for more rapid and efficient machines was emphasized. Part of the impetus behind the revival of dusts about 1915 was the need for lighter and more rapid equipment supplied by the dust rig. Airplane dusting began to appear on a commercial scale about 1930. A still more recent development is the vapo-dust, or fog spray, method with which both spray and dust are applied simultaneously. The air stream of the duster is used to carry the liquid spray from the nozzles to the plants in a fine mist, and at the same time water added to the air stream tends to deposit the dust particles on the foliage with less drift. Combinations of insecticides, fungicides, spreaders, and stickers may be used more effectively by this method.

FOLIAGE FUMIGANTS

The opportunity for the use of volatile materials as fungicides on foliage is limited to special cultures. In greenhouses, hotbeds, cold frames, and readily covered plant beds, the practical application of fumigants is sometimes feasible. Control of fruit or vegetable decays in storage rooms by gaseous materials is also a field largely unexplored.

Sulfur. Sublimated sulfur has long been used in greenhouses, where it is successful in controlling some fungus diseases, especially the powdery

mildews. The old empirical practice is that of placing powdered sulfur on heating pipes, where it gradually volatilizes and comes in contact with plant surfaces. Means have been devised to hasten this process by providing higher temperatures through electric hot plates.

Paradichlorobenzene. An outstanding example of recent origin in the use of fumigants is that originated by Angell *et al.* (1) in Australia for the control of downy mildew of tobacco (*Peronospora tabacina* Adams) in plant beds. They used benzene and toluene vapors in their experiments. In the United States, paradichlorobenzene has been found to be more suitable (23).

The standard treatment recommended in this country is as follows: A thin cotton cover, free of holes, is stretched tightly over the bed 10 to 15 in. above the ground. Crystals of paradichlorobenzene are scattered about sundown over the cover at the rate of 3 lb. per 100 sq. ft. in cool weather; the amount is reduced to 2 lb. in warm weather or $1\frac{1}{2}$ lb. if the cover is wet. To hold the vapors in the bed, a 60- to 70-thread muslin, weighing 4 to $4\frac{1}{2}$ oz. per sq. yd., somewhat larger than the bed, is stretched over the cotton cover and fastened down securely. The covers are removed in the morning before the sun has warmed up the bed. This treatment apparently has an eradicant effect and possibly deters sporulation by the fungus. It is therefore practical to apply the gas after the disease first becomes apparent. Treatment is carried out for three consecutive nights and semiweekly thereafter or as needed. Benzene vapor has been used successfully for the control of downy mildew of cabbage, *Peronospora parasitica* (Pers.) Fries, in cold frames in Mississippi (41). This fumigation measure is now replaced largely by the use of organic dusts.

FUNGICIDES APPLIED TO HARVESTED FRUITS AND VEGETABLES

There is a variety of situations in which the application of fungicides to perishable food products at or following harvest has come into successful use. The stem-end rot of watermelon (*Diplodia* spp.) is the result of infection through the stem, which is cut at harvest, opening wounded tissue to the pathogen. Since watermelons grown in southern states are shipped long distances, infected melons may be unfit for use by the time they reach the market. Copper sulfate paste applied to the cut surface of the stem as the melons are packed in the car was found to reduce this disease almost to nil (106). Applying benzoic acid to the cut stalk of pineapple fruit to control black rot, *Thielaviopsis paradoxa* (De Seyn.) Hoehn, is a process developed in Australia (30) and applied successfully in the United States (16). Sulfur dioxide gas released in storage rooms is used extensively to control the gray mold of grapes (*Botrytis* spp.) (33, 99)

The same principle was tried without much success by Hays (4) about 1850; he burned sulfur and introduced the fumes into the air surrounding potatoes in the storage bin to control late-blight rot.

Control of fruit rots in storage and transit by use of fungicide washes and impregnated wrappers has found extensive application. The blue mold (*Penicillium* spp.) of citrus fruits has been reduced greatly by the almost universal use in packing houses of an 8 to 10 per cent solution of borax at 120°F. as a fruit wash. This practice has been largely replaced by the use of wrappers impregnated with diphenyl, following initial experimentation and practice on citrus fruits in Palestine (50, 85, 107, 132, 133). Sodium ethyl mercurithiosalicylate, borax, sodium orthophenylphenol, and sodium metaborate have also been found to be effective when applied in solution to lemon fruits to prevent stem-end rot (*Phomopsis citri* Fawc. and *Diplodia natalensis* P. Evans) (44, 45, 142, 143). The same diseases in oranges have been controlled by use of 5 per cent thiourea or thioacetamide in solution as a dip or in wax emulsions; 8 per cent quinosol used as a dip is also effective (19, 20). Copper sulfate–impregnated wrappers with a minimum of 1.4 per cent metallic copper were found to be a preventive for rot of pear (*Botrytis* spp.) (24). When apples are rinsed previous to packing in the Pacific Northwest to remove spray residues, contamination of the rinse water and equipment by the blue-mold-decay organism (*Penicillium expansum* Link) may greatly increase the loss from the disease in storage. When sodium hypochlorite solution is added to the rinse to provide 0.4 per cent available chlorine and apples are exposed for 1 min., the amount of infection is markedly reduced (9). Chlorine and nitrogen trichloride are now used extensively in fruit and vegetable packing houses to decontaminate equipment, containers, and produce, and the latter material used as a gas in lemon packing houses is effective against storage decays (45).

SEED TREATMENT

It has already been pointed out that one of the early records of use of fungicidal materials for control of plant diseases was in connection with seed treatment. Salt-brine steeping of cereal seed arose as an empirical practice in the seventeenth century. Aucante (7) reported unsuccessful tests with mercuric chloride on grain seeds in 1755. In the same year Tillet (131) reported reduction in wheat bunt by the use of salt and lime as well as saltpeter on wheat seed. In 1761, Schulthess (116) suggested copper sulfate in place of salt brine. Prévost (104), in 1807, demonstrated the fungicidal and fungistatic effect of copper on the spores of the wheat-bunt organism and explained the basis of control of this disease by seed treat-

ment. Kühn elaborated upon the copper sulfate treatment of grains in his textbook in 1858 (71) and reported further experiments with the treatment in 1866 and 1873 (72, 73).

The copper sulfate treatment, while effective against wheat bunt, was not without disadvantages. One of these was a certain amount of injury to seed and seedlings. In the United States a common practice as early as 1862 (15) was to sprinkle the wet seed, after soaking in copper sulfate solution, with air-slaked lime. In 1873 (35) Dreisch in Germany reported addition of lime to copper sulfate solution to reduce toxicity of the latter to wheat seed. Another disadvantage was that loose smut of wheat was not controlled by copper sulfate. The distinction between bunt and loose smut and the fact that treatment with salt brine and lime did not control the latter were facts pointed out as early as 1755 by Tillet (131). It became evident to mycologists that the life history of loose smut was not identical with that of bunt. The fact that the loose-smut fungus penetrated the embryonic seed of barley was first suspected by Jensen (59) in Denmark (1888). It was later ascertained in Australia for loose smut of wheat by Maddox (87) (1896) and confirmed by Brefeld in Germany (1903) (17). The extent of the practice of seed treatment of wheat as well as its limitations in England in 1889 can be gauged from the remarks of Plowright (101), English authority on rusts and smuts:

> There is a certain point in connection with the reproduction of smut (*Ustilago segetum*) wherein it differs essentially from bunt (*Tilletia tritici*); it is this -that however carefully wheat may be dressed with cupric sulphate, arsenic, brine, lime, etc., while such dressing almost absolutely protects the crop from bunt, yet it has no appreciable effect upon smut. This fact is obvious to anyone residing in an agricultural district. The wheats are dressed for bunt on every well-managed farm, but they are much affected with smut as the barley and oat crops, which latter, never being affected with bunt, are never subjected to protective dressing.

Probably the inadequacy of copper sulfate was the reason for the interest of Jensen in Denmark in securing a better method. In 1888 (59) he described the hot-water treatment, which consisted in immersing seed in water at 132°F. for 15 min. and then spreading out in shallow layers to dry. Jensen's treatment was effective against loose smut of wheat and loose smut of barley as well as other smut diseases of small grains. The hot-water method was taken up promptly in the United States and compared with copper sulfate and other chemical treatments. Kellerman of Kansas (68), Arthur of Indiana, and Bolley (11) of North Dakota were each active in the field, beginning about 1889 and continuing through the next several years. Kellerman (66, 69) found hot water the most effective against wheat bunt. Although copper sulfate, copper acetate, copper

nitrate, potassium sulfide, and mercuric chloride were all about equal in fungicidal value with hot water, they all caused more or less seedling injury. Kellerman (67) also confirmed the observation of previous workers that copper sulfate solution was of no value in the control of loose smut of wheat. Bolley (11) confirmed the results of Kellerman with hot water, copper sulfate, and mercuric chloride. He was the first in the United States to use formaldehyde solution for treatment of grain and published encouraging results with the use of this material on oats in 1897 for loose smut and covered smut (14). Geuther (43) had published results on this chemical for grain-seed treatment in Germany in 1895. It became a widely used treatment for oats in spite of some tendency to injure seedlings and is still used on that crop.

At the close of the nineteenth century three methods of treating seeds of the small grains were in use, *i.e.*, hot water, copper sulfate, and formaldehyde. It had been ascertained that certain of these treatments were better adapted to some cereals than to others.

In 1902 Tubeuf (134) in Germany applied copper carbonate as a dust treatment of wheat seed for bunt. This apparently attracted little attention at the time. In 1917 Darnell-Smith (29) announced the treatment for use on wheat in Australia, and it soon became the most widely used treatment for wheat bunt. The use of organic mercury liquid treatment was reported in Germany in 1913 by Riehm (110). The search for new chemicals for seed treatment was stimulated by the growing recognition of the phytotoxic effects of copper sulfate and formaldehyde and of the cumbersome nature of the hot-water treatment. The organic mercury compounds offered greatest promise. During and after World War I a number of new seed-treatment compounds were introduced, most of them under patent processes and copyrighted names. After 1925 dust materials took decided precedence over liquid treatments.

In the decade before 1930 the field of seed protection began to receive greater attention. Up to that time most efforts in seed treatment had been directed toward organisms which were carried on or within the seed. The only extensive application of seed protection was that concerned with the control of onion smut. Thaxter (129) pointed out that the organism lived over in the soil and invaded the onion seedling only during a comparatively short period following seed germination. Working in Connecticut, he tried a considerable number of chemicals in dust form in the furrow with the seed and found a mixture of flowers of sulfur and air-slaked lime to be effective in reducing infection (1890). Sirrine and Stewart (121) conducted extensive field experiments in New York with a mixture of sulfur and lime and recommended its use in 1900. Selby (118) began experiments in Ohio in 1900 using formaldehyde solution, which

had been introduced as a seed disinfestant for oats by Bolley (14) in 1897. Selby found that a dilute solution applied in the soil with the seed was sufficient to protect the seedling from infection. Formaldehyde proved to be much superior to sulfur-lime dust for control of onion smut and has continued to be the standard practice up to the present date (118, 119). Attempts to apply formaldehyde adsorbed on dust carriers have been only partially successful. Arasan dust for onion-smut control is discussed later in this section (137).

It was emphasized by more and more investigators that damping-off and reduction in seedling stands were usually brought about by organisms in the soil flora rather than by organisms borne upon the seed. It has become necessary, therefore, to evaluate fungicides as seed protectants during germination of the seed and during the early stages of seedling growth. Here again relative phytotoxicity is as important as fungicidal value.

Organic mercury compounds were found to have some value as protectants, but cheaper and more effective materials were sought. Cuprous oxide, zinc oxide, and zinc sulfate were found to have high values for certain seeds. With the onset of World War II dire need of copper, zinc, and mercury for war materials gave a strong impetus to the search for nonmetallic seed protectants. A few outstanding materials were found which went rapidly into widespread use. One of these is chloranil, or tetrachloroquinone, now known as nabam, which is marketed under the trade name Spergon; another is tetramethylthiuram disulfide, known as thiram, which is marketed under the trade name Arasan. Still more recent are 2,3-dichloro-1,4-naphthoquinone, marketed under the trade name Phygon, and zinc 2,4,5-trichlorophenate, known as Dow Seed Fungicide (93, 138).

Types of Seed Treatment

Seed treatment may be divided into three categories depending upon the nature and purpose of the treatment. They are designated here as (*a*) seed disinfection, (*b*) seed disinfestation, and (*c*) seed protection. A given fungicidal treatment may serve in one or more of these categories.

Seed Disinfection. This refers to cases where treatment is directed toward eradication of the fungus which has infected the seed and is established within the seed coat or in more deep-seated tissues. The hot-water treatment devised by Jensen has been adapted to a number of organisms which infect the seed. Important examples are the organisms of loose smut of wheat, *Ustilago tritici* (Pers.) Rostr., and of barley; blackleg and black rot of crucifers; late blight of celery; stripe of barley (*Helminthosporium gramineum* Rabh.); and *Rhizoctonia* disease of tomato (8).

Chemicals are much more limited in this type of treatment than in seed disinfestation. Five per cent ethyl mercury phosphate (New Improved Ceresan) is effective against the barley-stripe fungus. One per cent ethyl mercury phosphate (Improved Semesan Jr.) and tetramethylthiuram disulfide (Arasan) are effective against the *Diplodia* seedling blight of corn (*Diplodia* spp.).

The bacterial-canker organism is eradicated in tomato seed if the macerated fruit pulp is allowed to ferment for 96 hr. at 20°C., owing to the increase in acid content of the matrix during fermentation. It has been found that freshly extracted seed may also be disinfected by soaking in 0.8 per cent acetic acid for 24 hr.

The field of seed disinfection is the most unsatisfactorily worked category of seed treatments. For many seed-borne organisms which infect seeds no adequate treatment has been found. Notable examples not now controlled by seed treatment are the organisms of the *Ascochyta* blights of garden pea, bacterial blights of common bean, and anthracnose of bean.

Seed Disinfestation. The seed treatments of cereal grains used in the seventeenth and eighteenth centuries were directed toward the smuts and were more or less effective against those in which the organism was present as chlamydospores on the surface of the seeds. Copper sulfate was effective, but it was also phytotoxic. When Bolley (14) initiated the use of formaldehyde, it became widely used, especially upon oats, and is still about the cheapest method which is also efficient. Copper carbonate and basic copper sulfate dusts overcame the disadvantage of copper sulfate solution, and the dust form was more easily applied than the liquid treatment. There were some disadvantages, one of which was the tendency of copper carbonate dust to impair the mechanical operation of the grain-seed drill. The organic mercury dusts and Arasan (tetramethylthiuram disulfide) are now widely used for disinfestation of seeds of small grains.

Among vegetable crops several standard practices are directed toward seed disinfestation. Treatment of cucurbit seeds with mercuric chloride solution is directed toward the anthracnose organism, *Colletotrichum lagenarium* (Pass.) Ell & Halst., and the angular-leaf-spot organism, *Pseudomonas lachrymans* (E. F. Sm. & Bryan) Carsner. Seeds of tomato, pepper and eggplant are treated with mercuric chloride solution or with ethyl mercuric phosphate in solution or as a dust for eradication of the organisms of bacterial spot, *Xanthomonas vesicatorium* (Doidge) Dows., and early blight.

Seed Protection. Seed protection is based on the principle of surrounding the seed and the young seedling with a fungicide which will prevent infection and damage by soil organisms, to which the plant is particularly vulnerable during this period of growth. Many of the or-

ganisms concerned are facultative parasites which do not affect the plants concerned except during such transient periods of susceptibility and at times when the environment is particularly favorable for infection and consequent disease development. Many vegetable crops suffer heavily at times from preemergence damping-off. Proper protection often brings striking results. Some field crops and many ornamental seeds are likewise greatly benefited. While cuprous oxide and zinc oxide were earlier used for certain vegetable seeds, they have now been largely supplanted by organic mercury dusts and by Spergon, Arasan, and Phygon. As may be expected, some seeds respond to one material better than to another, and in consequence no one compound serves as an over-all seed protectant. When a dust is required to serve as a seed disinfectant as well as a protectant, some dusts are preferable to others. For instance, 1 per cent ethyl mercury phosphate (Improved Semesan Jr.), chloranil (Spergon), and tetramethylthiuram disulfide (Arasan) are about equal as seed protectants for corn under average conditions, but if the seed is infected by *Diplodia* spp. and seed disinfection is also needed, Semesan Jr. and Arasan are far superior to Spergon, while Semesan Jr. has the disadvantage of being the most phytotoxic when mechanical injuries occur on the seeds.

An unusual case of seed protection occurs in connection with onion smut. The liquid formaldehyde treatment is cumbersome and requires special equipment. Dusts applied to the seeds have failed to be sufficiently protective to warrant their use. One reason for this is the fact that the seedling must be protected between the root and the soil surface, while the seed coat, usually carried above ground by the expanding cotyledon, does not carry enough dust to disinfest the soil in the critical zone. Formaldehyde applied in solution soon volatilizes and thus presumably affects a sufficient zone to be effective. By increasing the amount of Arasan adhering to the seed the same result is accomplished. The method devised is to coat the seed first with Methocel, a cellulose acetate sticker, applied in dilute solution. By agitating such seed with the dust, many times the usual amount can be made to adhere, with the result that individual seeds become pellets containing an outer zone of Arasan (137).

Common Recommendations for Seed Treatment

Because of the varied requirements for disinfection, disinfestation, and protection, because of the regional variation in disease importance, and because of continual production of new materials by research laboratories, recommendations for commercial use are continually changing. Some of the procedures and materials listed in Table 3 may therefore be obsolete sooner or later. The list is given here to illustrate the formulae and materials in current use in 1950.

TABLE 3. SOME COMMON SEED TREATMENTS

Crop	Material	Method	Rate, etc.	Diseases concerned	Type of effect[a]
Wheat	New Improved Ceresan[b]	Dust	½ oz. per bu.	Bunt and flag smut	2
				Seedling blight	2, 3
	Ceresan M[c]	Slurry	1 lb. per gal. to 32 bu.	Bunt and flag smut	2
				Seedling blight	2, 3
	Copper carbonate	Dust	2 oz. per bu.	Bunt and flag smut	2
	Hot water	Soak	Unheated water, 4–6 hr.; at 120F., 2 min.; at 129°, 10 min.	Loose smut	1
Oats	New Improved Ceresan	Dust	½ oz. per bu.	Loose and covered smuts	2
				Seedling blight	3
	Ceresan M	Slurry	1½ lb. per gal. to 32 bu.	Loose and covered smuts	2, 3
				Seedling blights	3
	Formaldehyde[d]	Spray	1 pt. to 1 pt. water sprayed on 50 bu.	Loose and covered smuts	2
Barley	New Improved Ceresan	Dust	½ oz. per bu.	Covered and black loose smuts	2
				Stripe	1
				Seedling blight	3
	Ceresan M	Slurry	1½ lb. per gal. to 32 bu.	Covered and black loose smuts	2
				Stripe	1
				Seedling blight	3
	Hot water	Soak	Unheated water, 12 hr.; at 127°F., 13 min.	Loose smut	1
Flax	New Improved Ceresan	Dust	1½ oz. per bu.	Seedling blights	3
Corn	Arasan SF[e]	Slurry	1 lb. per gal. to 30 bu.	Seedling blights	3
				Diplodia rot	1
Sorghum	New Improved Ceresan	Dust	½ oz. per bu.	Smut and seedling blight	2, 3
	Arasan[f]	Dust	1½ oz. per bu.	Smut and seedling blight	2, 3
	Spergon[g]	Dust	1½ oz. per bu.	Smut and seedling blight	2, 3
	Phygon[h]	Dust	1 oz. per bu.	Smut and seedling blight	2, 3
Pea	Spergon	Dust	2 oz. per bu.	Damping-off	3
Lima bean	Spergon	Dust	2 oz. per bu.	Damping-off	3
Spinach	Arasan	Dust	0.25%	Damping-off	3
	Phygon	Dust	0.25%	Damping-off	3
Beet	Arasan	Dust	0.50%	Damping-off	3
	Phygon	Dust	1.00%	Damping-off	3
Carrot	Arasan	Dust	0.75%	Damping-off	3
	Hot water	Soak	126°F., 10 min.	Bacterial blight	1, 2
Tomato, pepper, eggplant	New Improved Ceresan	Dust	0.50%	Early blight and bacterial spot	2
	Hot water	Soak	125°F., 30 min.	*Rhizoctonia* damping-off	1
Cucurbits	Mercuric chloride	Soak	1–1,000, 5 min.	Anthracnose and angular leaf spot	2
	Arasan	Dust	0.3%	Damping-off	3
	Spergon	Dust	0.3%	Damping-off	3
Crucifers	Hot water	Soak	122°F., 30 min.	Blackleg, black rot, black leaf spot, ring spot	1
	Arasan	Dust	0.50%	Damping-off	3

[a] 1 = disinfection; 2 = disinfestation; 3 = protection.
[b] 5 per cent ethyl mercury phosphate.
[c] 7.7 per cent ethyl mercury *p*-toluenesulfonanilide.
[d] 40 per cent solution in water.
[e] 75 per cent tetramethylthiuram disulfide.
[f] 50 per cent tetramethylthiuram disulfide.
[g] Chloranil, or tetrachloroquinone.
[h] 2,3-Dichloro-1,4-naphthoquinone.

Seed-treatment Equipment

For four decades after 1890 treatment of seeds and tubers was practiced primarily by the individual grower. For small-scale operation relatively simple homemade devices for application of liquids or dusts were sufficient. When hot-water treatment was used, more precise methods were necessary. Large-scale treatment machinery was developed as the practices expanded. Potato tubers are bulky, and up to 20 bushels per acre are needed for planting. Along with shortening of the 2-hr. soak, large-scale equipment was devised to provide a continuous flow of tubers through the treatment fluid. This was particularly useful in the application of the hot formaldehyde treatment described below under Tuber Treatment.

When seeds are treated in small volume, dusts are usually applied in a revolving drum, with internal baffles, mounted on an eccentric axis. The Minnesota seed-grain treater is a convenient small-scale device in which the seed and the dust are mixed by dropping through a vertical chute containing baffles. During recent years volume treatment has been accomplished by large-scale equipment at warehouses or by combination seed-cleaning and- treating equipment transported upon a motor truck.

One of the difficulties of large-scale application of dust treatment to seeds at the source or at distribution points is the inconvenience and danger to operators of continual operation in atmosphere to which some of the dust inevitably escapes. Most dusts in use cause some irritation to the skin or the mucous membrane of the respiratory tract. Much of this can be avoided by adequate exhaustion of air by blowers from the vicinity of the treating equipment. The slurry method of seed treatment is a device designed to circumvent the disadvantages of dust treatment. The dust is manufactured with a wettable carrier. The dust in the machine is contained in water in a tank through which seed is drawn in small buckets on an endless belt. As the seed leaves the dust bath, it is not noticeably moist and since a drying process is unnecessary, sacking can be done directly from the treater. The chief advantages of this equipment are that it eliminates dust in the atmosphere and it provides more uniform and more accurate application.

Another recent development in seed treatment has arisen from the need to increase the load of dust applied over what would ordinarily adhere. A sticker may be applied to the seed to increase the dust load. By agitating such seed in a rapidly revolving drum, uniform layers of dust on the exterior may be secured. This is commonly referred to as "pelleting." The first commercial application of this principle was the use of Methocel, a soluble cellulose acetate sticker, on onion seed to increase the load of

Arasan. Onion seed can thus be pelleted with sufficient protectant to control infection by the onion-smut organism in the soil.

TUBER TREATMENT

The organisms which incite potato scab and black scurf are carried on tubers. The first work on treatment of seed tubers was published by Bolley (12, 13) in North Dakota in 1891. He recommended soaking tubers in 1–1,000 mercuric chloride for $1\frac{1}{2}$ hr. This became one of the standard formulae used during the following four decades. In 1897 Arthur (6) in Indiana reported on the use of formaldehyde solution, and it also became a standard treatment at a dilution of 1 part of concentrated formaldehyde solution to 240 parts of water for a period of 2 hr. A considerable handicap in the use of these treatments was the length of the period of soak. About 1920 two lines of development led to shortening of the treatments. Melhus (92) in Iowa worked out the hot formaldehyde treatment, wherein, with a stronger solution (1–120) kept at 50°C., the interval of treatment was reduced from 2 hr. to 4 min. The mercuric chloride treatment was shortened for $1\frac{1}{2}$ hr. to 5 min. by addition of 1 per cent of hydrochloric acid to the solution (26, 75). Both of these treatments proved to be more liable to cause injury to the tubers, particularly if the latter had passed the dormant period and were sprouting. An organic mercury liquid treatment, Semesan Bel, composed of 12 per cent hydroxymercurinitrophenol and 2 per cent hydroxymercurichlorophenol, used at 1 lb. to $7\frac{1}{2}$ gal. of water requires only an instantaneous dip.

While it is possible to reduce, if not eliminate, the scab and scurf organisms on the surface of the tubers, the value of tuber treatments has been seriously questioned in many areas, and in many states treatment is no longer recommended. Lack of success in the control of the diseases concerned is commonly due to the widespread infestation of soil by the pathogens. Another reason may be found in the relatively recent recognition of the fact that sclerotia of *Pellicularia* on potato tubers are in a large percentage of cases those of strains of the organism which are very weakly pathogenic or even nonpathogenic (114, 122). In the British Isles the treatment of seed tubers to prevent infection by the late-blight organism is sometimes used. Greeves (47) showed that if tubers dug while infected vines were still bearing fruiting bodies were soaked immediately with mercuric chloride (1–1,000, 90 min.) or dipped in an organic mercury preparation, the percentage of rot in storage was reduced from around 70 to 2. Cunningham and Reinking (27) reduced seed-piece decay by use of the Semesan Bel dip or cuprous oxide dip (1 lb. to 30 gal. water).

BULB TREATMENT

Bulbs of flowering plants, particularly those of narcissus and gladiolus, are commonly treated for nematodes and for bulb-rotting fungi. For nematodes a combination of hot water and formaldehyde is used (21). For the control of *Fusarium* bulb rots, Arasan dust is commonly applied. Eradication of *Colletotrichum lilii* Plak. from bulbs of Easter lily was not satisfactory when dips or dusts of mercuric chloride, borax, basic copper sulfate, and calcium hypochlorite were used but was accomplished by soaking for 48 hr. in 1-2,000 solution of puratized N5E (10 per cent phenyl mercuri triethanolammonium lactate). This treatment did not protect the bulbs from reinfection by the same organism in the soil. Arasan, while of no value as an eradicant of *C. lilii*, did prove to be an excellent protectant. Therefore, a soak with N5E followed by application of Arasan dust was found to be the most effective combination of treatments (77–79).

The use of fungicides on bulbs has not been the subject of much experimentation. It may be expected that with the development of new fungicidal materials this field of disease control will see greater improvements in the future.

TREATMENT OF OTHER PLANT PARTS

Sweet-potato roots to be used for production of sprouts are usually treated before bedding by soaking for 10 min. in borax solution (6 lb. in 30 gal. of water) or in mercuric chloride solution (1 oz. in 8 gal. of water). Various materials have been used to treat sweet-potato sprouts before setting into the field. Most-successful treatments include Semesan Bel and mercuric chloride (1 oz. in 8 gal. of water).

In Louisiana a hot-water treatment (52°C., 20 min.) of sugar-cane cuttings has been developed for the control of red rot (*Physalospora tucumanensis* Speg.) (38). In India, cane smut (*Ustilago scitaminea* Syd.) is controlled by dipping cuttings in 1-1,000 mercuric chloride or 1-100 formaldehyde for 5 min. (81). In South Africa cuttings are dipped in an organic mercurial solution to control infection by *Ceratostomella paradoxa* (86). Hot-water treatment of roots of *Aloe variegata* L. and rhizomes of calla lily (50°C., 1 hr.) has also been successful (31). According to Staniland (124), mint rust (*Puccinia menthae* Pers.) can be controlled by treating runners before forcing in water at 43°C. for 10 min.

In Hawaii heart rot of pineapple plants is incited by three species of *Phytophthora*. Mehrlich (91), after testing 67 formulae of 22 liquid and 12 dry fungicides, found that best results were obtained by completely immersing planting material in 1-0.7 3 Bordeaux mixture.

SOIL TREATMENT

The removal of plant pathogens from the soil by means of chemicals and by means of heat are procedures in common use in greenhouses, hotbeds, cold frames, and outdoor seedbeds. Heating of soil is an old method used to kill weed seeds, insects, fungi, bacteria, and nematodes. The most common method of heat treatment is the use of steam under pressure (5, 61, 62, 120).

A common method of applying steam, especially in open seedbeds, is by use of an inverted metal pan. In greenhouses steam is commonly introduced through the tile drainage system, whereby it is allowed to permeate slowly into the moistened soil, which is covered with canvas to retain the heat. Introduction of steam through a perforated rake or harrow is less widely used.

Complete sterilization of the soil is not usually necessary, but formulae of time and temperature are gauged to rid the soil of nematodes and pathogenic fungi and bacteria. Steaming of soil to the point necessary to accomplish these objectives usually results in the accumulation of toxic materials, chiefly free ammonia, which injure sensitive plants and seeds if the latter are placed in the soil immediately. A period of several days is usually allowed to elapse, during which time contamination of the soil with microorganisms tends to break down injurious materials. Ordinarily the organisms which reinfest the soil are not pathogenic, but it is highly important that steamed soil be protected from contamination with pathogenic organisms from nonsteamed soil. Soil only mildly steamed without pressure is often more satisfactory because of the lower risk of toxicity to plants (61, 62).

Devices for pasteurizing soil by electric heaters have been made (54, 97). These are useful for special purposes but are not generally economical for large-scale commercial use.

Various chemical treatments of soil have been developed. The oldest and probably still most commonly used, where steaming is not feasible, is the formaldehyde drench. This consists in soaking the soil in beds to a depth of about 6 in. with a formaldehyde solution made up at the rate of 1 gal. of concentrated solution (37 to 40 per cent formaldehyde) to 50 gal. of water. The soil is covered with canvas for 1 or 2 days to retain the gas. The soil is not used until all traces of formaldehyde have disappeared. The application of formaldehyde with steam, when applied through drainage tile, has been found to be useful in certain cases, and it has the advantage of reducing the time period ordinarily required for steam alone (10). Vinegar is satisfactory as a mild treatment of soil in some cases

(34). For forest-tree nurseries Riker *et al.* (111) secured relatively good results with Arasan and with Barbak C (8 per cent mercuric phenyl cyanamide and 2.5 per cent cadmium oxide) supplemented by mercurous chloride.

Soil fumigants other than formaldehyde have been used to some extent, but their acceptance has been less universal. Chloropicrin is useful both as a fungicide and as a larvicide in greenhouse soils. It is injected into the soil 3 to 6 in. in holes 9 to 12 in. apart, and the soil is covered for 48 hr. with impervious cloth or paper. An interval of a week or more is required before sowing or planting. About 1942 dichloropropene-dichloropropane mixture, commonly known as DD, was introduced for greenhouse as well as for large-scale outdoor use (84). It is successful in the control of nematodes but appears to have little or no bactericidal or fungicidal value in the soil. It can be applied to the soil at the time of plowing. Other chemicals which have larvicidal value but little or no fungicidal value in the soil are ethylene dichloride, ethylene dibromide, and methyl bromide (96). In general, fumigants are most effective in wet soils and are retained longest in wet, cold soil (82).

WOOD PRESERVATION

As already indicated, several processes of wood preservation were devised before 1850. The immersion of timber in mercuric chloride solution, known as Kyanizing, was patented by Kyan in 1832. The use of coal-tar creosote as a perservative was patented by Moll in 1836, and a pressure process for application of this material was patented by Bethell in 1838. Margary secured a patent on the use of copper sulfate as a preservative in 1837, and Burnett patented the use of zinc chloride in 1838. Procedures in vogue at the turn of the century are described by Schrenk (115).

While phytotoxicity is not a factor in wood preservation, there are various requirements of a wood preservative, some of which do not apply to a foliage or a seed fungicide. Chief of these are:

1. Permanent value over a period of many years.
2. Capability of being injected readily.
3. Low hazard to workmen during processing.
4. Low inflammability.
5. Noncorrosiveness to metal fittings, nails, bolts, etc.
6. Lack of color and odor for finishing woods.

Coal-tar creosote is the most extensively used preservative. Zinc chloride is the most widely used water-soluble material. Creosote combined with petroleum oils is used extensively, and creosote combined with zinc chloride in lesser amount. Pentachlorophenol is also used extensively for

certain purposes. Approximately half of the timber treated is used for railroad ties; about one-fourth is used for poles; construction timbers are next in volume; piles rank fourth.

A number of proprietary compounds are recommended for use as wood preservatives. Among these are Ac-zol, containing copper sulfate (or oxide), zinc oxide, phenol, and ammonia; Ascu, containing copper arsenic and chromium salt; Basilit, containing fluoride-phenol; Bruce preservative, containing beta-naphthol; carbolineums, or anthracene oils, under numerous trade names; Celeure, containing potassium dichromate, copper sulfate, and acetic acid; Chemonite, containing copper arsenite; Permatol, containing pentachlorophenol and related phenols; Wolman salts, containing fluoride-phenol mixtures.

The engineering phases of wood preservation are very involved, and the expense of equipment requires volume processing in order for the procedure to be economically feasible. Treated wood up to the present is therefore confined largely to a few large users, particularly the railroads. Treated wood for construction purposes is still unavailable to the small user. The entire subject is thoroughly covered in "Wood Preservation" by Hunt and Garratt (58).

Various devices for home application of wood preservatives have been developed. Since water-soluble materials move more rapidly through sapwood than through heartwood, it is possible to treat green saplings or small trees inexpensively and greatly prolong their use as poles or posts. Newly cut green saplings or trees can be stood upright in a container of preservative, and the chemical will rise in the sap stream and disperse throughout the wood. Chromated zinc chloride is more effective than zinc chloride and less corrosive on wire and nails than copper sulfate.

Another method is that known as the tire-tube method for treating posts. The bark is peeled from the large end of the green post for 4 to 6 in.; grease is applied, and a section of inner tube 2 to $2\frac{1}{2}$ ft. long is slipped over the end and bound. The post is then laid on a rack with the large end about $1\frac{1}{2}$ ft. higher than the small end. The loose end of the tire is fastened in an upright position and a measured amount of preservative added. Zinc chloride or chromated zinc chloride diluted to a 10 per cent solution is used. A post 8 ft. long and with an average diameter of 8 in. requires 3 gal. of preservative. Dosage tables for various sizes and lengths of posts can be secured. When the liquid has been completely absorbed, the tire is removed and the post stored for future use.

Blue stain is due to a number of species of *Ceratostomella*, which, although they contribute little to the cause of wood decay, may cause considerable damage to wood during storage or transportation by the massing of the dark-colored hyphae on or near the surface of cut lumber during the first

3 or 4 weeks of seasoning. Ethyl mercury chloride was used extensively as a dip to control this trouble. Ethyl mercury oleate in emulsified oil miscible with water was developed next. A still more recent material, known under the copyrighted name "New Improved Lignasan," contains ethyl mercury phosphate as the fungicide. It is applied in water solution and is noncorrosive to iron and steel. Other proprietary compounds recommended are Dowicide H and Permatox 108. (49, 136).

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CHAPTER 17

DISEASE CONTROL THROUGH HOST RESISTANCE

The observation that varieties of economic plants differ in susceptibility to disease was probably not new when it was recorded by Theophrastus (371–286 B.C.). We know little of what use was made of the fact, deliberately, in the amelioration of cultivated varieties in succeeding centuries, but probably many varieties were discarded empirically because of extreme susceptibility to one or another disease. Beginning in the early part of the nineteenth century, an increasing number of references to relative resistance of varieties was recorded. Thomas Andrew Knight, a well-known English plant breeder, who worked in the first half of that century, noted differences in resistance of wheat varieties to rust. In 1853, Anderson in England noted that certain varieties of turnip were more resistant than others to clubroot. Berkeley in 1851 in the original description of onion smudge observed that white varieties were seriously affected while colored varieties growing alongside were free from the disease.

Following the recognition of the causal relation of fungi to disease development, the possibility of control through disease resistance received consideration in a few instances, but these may be looked upon as exceptional rather than usual cases. The losses in the potato crop from late blight, which continued in Europe and America after the 1840's, stimulated amateur breeders in both continents to improve upon the current varieties by hybridization with recently introduced stocks from South America. As already noted under the discussion of this disease (p. 206), the Magnum Bonum variety developed in England and introduced in 1876 showed marked resistance to late blight, but by the end of the century its record was less widely satisfactory than had been hoped. In 1903, Sorauer (19), in Germany, wrote of this variety as follows:

> Among a few examples toward which resistance is shown by the plant, it may be noted that the rot of potato caused by *Phytophthora infestans*, our most common potato disease, occurs in a wholly various manner on different varieties under the same system of culture, planted at the same time. According to my earlier investigations, in general the white, smooth-skinned, early varieties with thin corky layers, with less starch and higher in protein, suffer more than the later, rough-skinned, and for the most part red, varieties. Likewise it is shown that fresh manuring delays maturity and therewith the duration of younger susceptible tendencies is increased. At the same time it is expected, and established through recent observations in practice that the resistance of some varieties always varies according to cultural conditions. Varieties, such as Magnum Bonum, which are many times observed as peculiarly fungus resistant, retain it only so long as they are

cultivated in the light types of soil suitable for them, but show up as very weak and are inclined to rot when they are grown in heavy, moist soil.

When Jones (11) made a collection of varieties in Europe in the early part of the current century and together with Stuart (20) tested them for resistance to late blight, he found them to be more resistant as a whole than varieties in current use in America.

The impact of the rapid development and extreme destructiveness of downy mildew of grape in France in the 1880's brought to the attention of Millardet and other French investigators the fact that American varieties of grape, which had been introduced because of their resistance to the root louse were also comparatively much more resistant to downy mildew. Millardet initiated hybridization of American and European grapes with the objective of introducing thereby the resistant characteristics of American grapes into European wine grapes.

In 1883 Little reported in the results of a questionnaire sent to English farmers that wheat growers in that country generally regarded Red and Rivet varieties resistant to rust. With the expansion of wheat growing in Australia in the last two decades of the nineteenth century, black stem rust became an important disease. Farrar was an active wheat breeder in New South Wales at that time. Associated with him was the American plant pathologist N. A. Cobb. The latter carried out very important studies on the relative resistance of wheat varieties and upon the nature of the differences between resistant and susceptible varieties. This was the beginning of an important wheat-breeding program, in which improvement in rust resistance was a major part. Only a few years later Carleton and others in America began the collection of wheat varieties in Europe as the basis of breeding programs which still continue and in which resistance has become of increasing importance. In England, Biffen began a similar program shortly before 1900.

With the rediscovery of Mendel's laws of heredity in 1900, the science of genetics had an increasing influence upon plant breeding. Biffen (2, 3) was the first to apply Mendel's laws to the inheritance of disease resistance. He crossed the wheat variety Rivet with the variety Michigan Bronze. The former was highly resistant to stripe rust, *Puccinia glumarum* (Schm.) Erikss. & Henn.; the latter was susceptible. The F_2 progeny segregated in the ratio of three susceptible to one resistant, while the F_3 families were grouped into approximately one-fourth true-breeding resistant lines, one-fourth true-breeding susceptible lines, and one-half segregating lines.

It is interesting to note the skepticism with which the pronouncement of Biffen was received by plant pathologists. Butler (5) in India pointed out promptly that resistance in wheat for a given rust applies only to a

particular variety in a particular locality and does not necessarily hold when the variety is transferred to another locality. Evans (8), in South Africa, carried on studies with resistance to *Puccinia graminis*. He used as one parent Bob's Rust Proof, a variety from Australia which remained consistently free from the black stem rust at Pretoria, although it rusted badly in some other parts of the Transvaal. A local variety, Wol Koren, was used as a susceptible parent. Susceptibility was dominant in the F_1; and, in fact, the hybrid plants were more severely affected than those of the susceptible parent line. Furthermore, the rust from the hybrid plants was infectious to the resistant parent line, while rust from plants of the susceptible parent line was not. Evans was also inclined to disparage the practical value of breeding for resistance, since it was thought that resistance would decline in new varieties as rapidly as they could be produced. In 1911, Nilsson-Ehle (13), in Sweden, reported results from crosses between lines resistant and susceptible to *P. glumarum*. The parents were different from those used by Biffen, and the data indicated polygenic rather than monogenic inheritance.

Up to 1911, therefore, there was still much confusion as to the stability and inheritance of rust resistance. This situation is readily explained now by the following facts: The extent of physiologic specialization of rusts was not fully understood. The influence of environment on the expression of resistance was overemphasized by some and unduly minimized by others. The Ward theory of bridging hosts (p. 410) was accepted by many, and this tended to overemphasize the role of the semiresistant host in increasing the virulence of a given rust collection. Biffen was not discouraged by doubts of other investigators, for he pointed out that at least in certain varieties the resistant character remained stable in England and that the Rivet variety, which was one of the oldest in cultivation, had long been known to be resistant to yellow rust and continued to remain so.

While rust resistance in wheat was a subject of controversy in Europe during the first decade of the current century, some notable advances in the control of disease through resistance were being made in America. The *Fusarium* wilt of cotton, *Fusarium oxysporum* f. *vasinfectum* (Atk.) Snyder & Hansen, had crippled the growing of the crop in certain sections of southeastern United States. The *Fusarium* wilts of watermelon, *F. oxysporum* f. *niveum* (E. F. Sm.) Snyder & Hansen, and cowpea, *F. oxysporum* f. *tracheiphilum* (E. F. Sm.) Snyder & Hansen, were also recognized as threats to the respective crops before 1900. Orton then initiated a study of these three diseases about 1900 and in each case made substantial progress in their control through disease resistance. In cotton he

found certain locally selected strains more resistant than others and showed that resistance was improved by selection of survivors within varieties grown upon naturally infested soil. This early work was the foundation of breeding programs which followed in various sections and resulted in numerous resistant varieties in use at the present time. In watermelon he found all commercial varieties very susceptible, but the African citron, a member of the same species as watermelon, was highly resistant. From a cross between watermelon and citron he selected a highly resistant variety, Conqueror. Although not widely used because of certain limitations in horticultural characteristics, this variety served as a parent in later breeding programs. In cowpea, a naturally resistant variety, Iron, was discovered. The *Fusarium* wilts of flax, *F. oxysporum* f. *lini* (Bolley), Snyder & Hansen, and of tomato, *F. oxysporum* f. *lycopersici* (Sacc.) Snyder & Hansen, became important in the United States soon after 1900. Bolley (4) described flax wilt in the Red River valley of North Dakota and observed healthy survivors in severely diseased crops. By selection, he secured highly resistant strains, one of the first to be released being the variety Bison. Essary (7) in Tennessee by similar methods secured wilt-resistant strains of tomato. In 1909, Jones and Gilman (12) began selection of cabbage in Wisconsin, which led to the release of a highly resistant variety, Wisconsin Hollander, in 1916.

In each of these studies of resistance to a vascular *Fusarium* disease remarkably rapid improvement was secured by selection of resistant survivors within horticultural or agronomic varieties, except in cowpea, where a naturally resistant variety was found, and in watermelon, where an outcross was first made. Little or no attention was given to the nature of inheritance of the resistant character. The fact that the character was inherited was not questioned, but it was not claimed that it behaved as a unit character in any of the cases. Furthermore it was implied by direct inference or otherwise that resistance was not completely fixed, that continuous selection was advisable, and that environment might be expected to influence the degree of resistance in any one situation as compared with another. It is worthy of note that, some 30 years later, many of the varieties are still used commercially as a means of control of the respective diseases, while others have served as parents in crosses designed to improve them in horticultural or agronomic characteristics.

PRESENT IMPORTANCE

Beginning about 1920 host resistance in the case of many plant diseases received rapidly increasing attention in Europe and America. Many of the important diseases of numerous economic crops are controlled by this

means. Among the diseases already treated in detail in previous chapters the following are listed as illustrative of those in which host resistance is relied upon as the chief remedy:

Clubroot of turnip and rutabaga.
Black wart of potato.
Cabbage yellows.
Fusarium wilt of tomato.
Onion smudge.
Black stem rust.
Flax rust.
Common mosaic of bean.
Cucumber mosaic.
Curly top of sugar beet.

Other cases not treated in detail which should be included are pea wilt (21, 24), pea near-wilt (10), *Cercospora* leaf spot of sugar beet (6), and asparagus rust (14).

Among many of the diseases treated in detail, host resistance, while not the major control remedy, is one of secondary importance in that some varieties are less susceptible than others or certain varieties are resistant to a number of physiologic races of the pathogen. In others breeding programs are just beginning to bring out resistant varieties. Many such cases will be found in the literature in addition to those treated here:

Bacterial blights of bean.
Southern wilt of tobacco.
Ring rot of potato.
Bacterial wilt of curcurbits.
Angular leaf spot of cotton.
Fire blight of apple and pear.
Late blight of potato.
Downy mildew of grape.
Downy mildew of cucumber and of cantaloupe.
Neck rot of onion.
Bean anthracnose.
Brown rot of peach.
Apple rust.
Oat smuts.
Wheat bunt.
Tobacco mosaic.
Spotted wilt.

Summaries of progress in development of disease-resistant crop plants are available (1, 18, 22, 26).

INHERITANCE OF DISEASE RESISTANCE

There are many cases of disease resistance in which the genetic pattern of inheritance has been worked out. Resistance is sometimes controlled by a single gene pair. An example already discussed is cabbage yellows, where in one type of resistance the latter is dominant over susceptibility. Many cases similar to this are now known to exist, such as resistance to pea wilt (21) and resistance to near-wilt of pea (10). In each of these cases the organism is relatively stable pathogenetically, although isolates of both vary somewhat in virulence. In angular leaf spot of cotton two gene pairs for resistance occur. In onion smudge the interaction of three gene pairs is involved in the determination of whether or not a bulb is highly resistant, intermediate in resistance, or highly susceptible. In many cases resistance behaves as a quantitative character and is polygenic in its inheritance. This is the case with root rot of tobacco and type B resistance to cabbage yellows.

When the organism consists of well-defined physiologic races, as in black stem rust, wheat bunt, and pea wilts (Fig. 189), specific genes for resistance to specific races usually obtain. Thus, for several races of *Puccinia graminis tritici* it has been shown that resistance is in each case controlled by different gene pairs. In the case of bean anthracnose resistance to races *alpha*, *beta*, and *gamma* was shown to be controlled by independent gene pairs. When analysis of resistance to a given pathogen is carried out, it is important to know whether the isolate of the organism is a stable race, whether it is varying by mutation or heterocaryosis, and whether it consists of more than one distinct race.

Resistance to a given race may be phenotypically similar but genotypically distinct in different host varieties. In bean mosaic two types of resistance are known; under most conditions these are similar in appearance but are inherited differently. These two types, the Corbett Refugee type and the Robust type, have been shown to be controlled by the interaction of two gene pairs. Moreover, when the two types, though highly resistant, are crossed, the F_2 generation segregates into resistant and susceptible classes. Susceptibility is dominant, and when a susceptible plant is crossed with Robust, the F_1 is susceptible. However, when the susceptible is crossed with the Corbett Refugee type, the F_1 is resistant, because the latter parent has a gene which is a dominant inhibitor of the susceptible gene.

In resistance to race 3 of powdery mildew of barley nine distinct genes for resistance occur in different barley varieties. Some are independent; some are loosely linked; but each case of resistance is phenotypically similar to each other case.

FIG. 189. Resistance to pea wilt and near wilt incited, respectively, by race 1 and race 2 of *Fusarium oxysporum* f. *pisi* (Linford) Snyder & Hansen. *A*, on soil infested with race 1, the susceptible variety (left) has succumbed, while the variety on the right is homozygous for resistance to race 1. It is, however, susceptible to race 2. *B*, soil infested with race 1 and race 2. The variety on the left is Wisconsin Perfection, resistant to race 1 but susceptible to race 2. The variety on the right, Delwiche Commando, is homozygous for the two dominant genes (*Fw* and *Fnw*) for resistance to race 1 and race 2, respectively. (*B after Hare, Walker, and Delwiche.*)

Sometimes two types of resistance are similar under one set of conditions but not under another. This brings us to a consideration of environment in relation to the expression of disease resistance.

ENVIRONMENT IN RELATION TO RESISTANCE

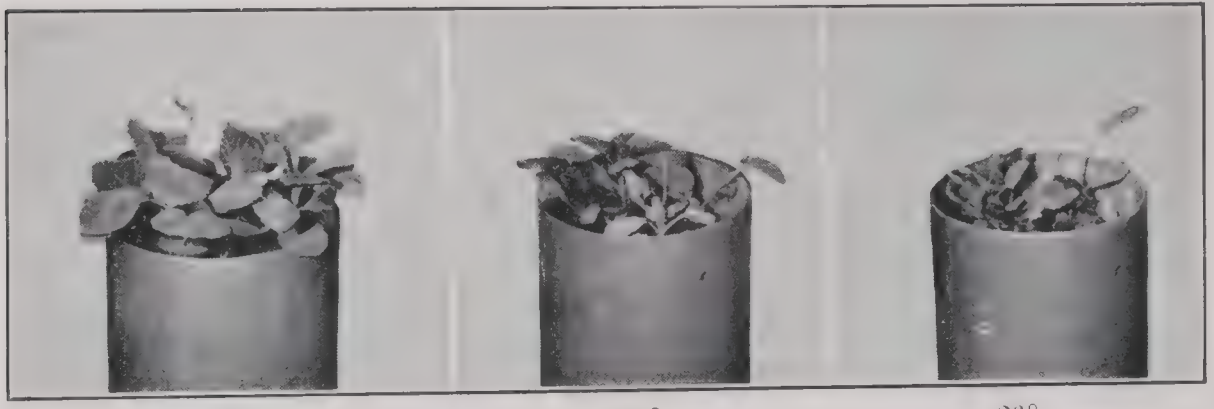
Variation in the expression of resistance has been shown to be brought about by difference in the pathogenicity of the organism and by difference

FIG. 190. Types of resistance to cabbage yellows. The variety on the left (Wisconsin Ballhead) selected from the susceptible variety in the center (Danish Ballhead) is homozygous for the single gene for type A resistance. The variety on the right (Wisconsin Hollander), also selected from Danish Ballhead, is resistant under moderately favorable environment. The latter resistance (type B) is polygenic and becomes unstable in phenotypic expression when the soil temperature is unusually high (compare with Fig. 191).

in the genes governing resistance. Environment has a part in the final picture by its effect on disease development. As a general rule a resistant character which is controlled by a single gene pair, and thus acts as a qualitative character, is stable in its expression over a fairly wide range of environment. In carrying out a breeding program with this type of resistance or in studying the inheritance of resistance, segregating progenies can usually be divided into distinct discontinuous classes. This is the case with pea wilt (21, 24), type A resistance to yellows in cabbage, re-

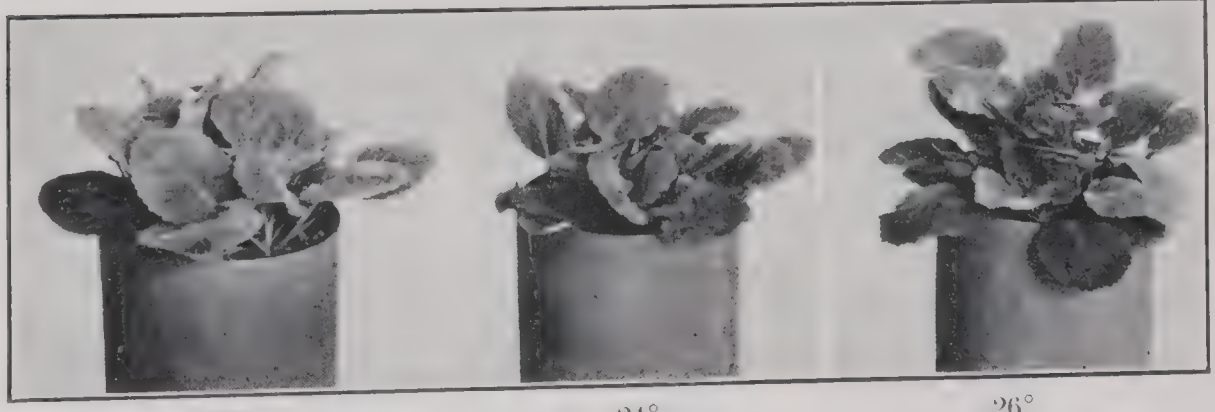
22° 24° 26°

SUSCEPTIBLE



22° 24° 26°

TYPE B, RESISTANT



22° 24° 26°

TYPE A, RESISTANT

FIG. 191. The same varieties shown in Fig. 190 grown in infested soil at the constant soil temperatures indicated. Note that type A resistance is stable up to 26°C., while type B resistance is almost completely suppressed in expression at 24° (see Fig. 192).

sistance to given races of rusts and smuts, and in many other cases. If, however, the environment is at one or another edge of an optimum range, disease classes may become obscure. Thus, it was shown that under high

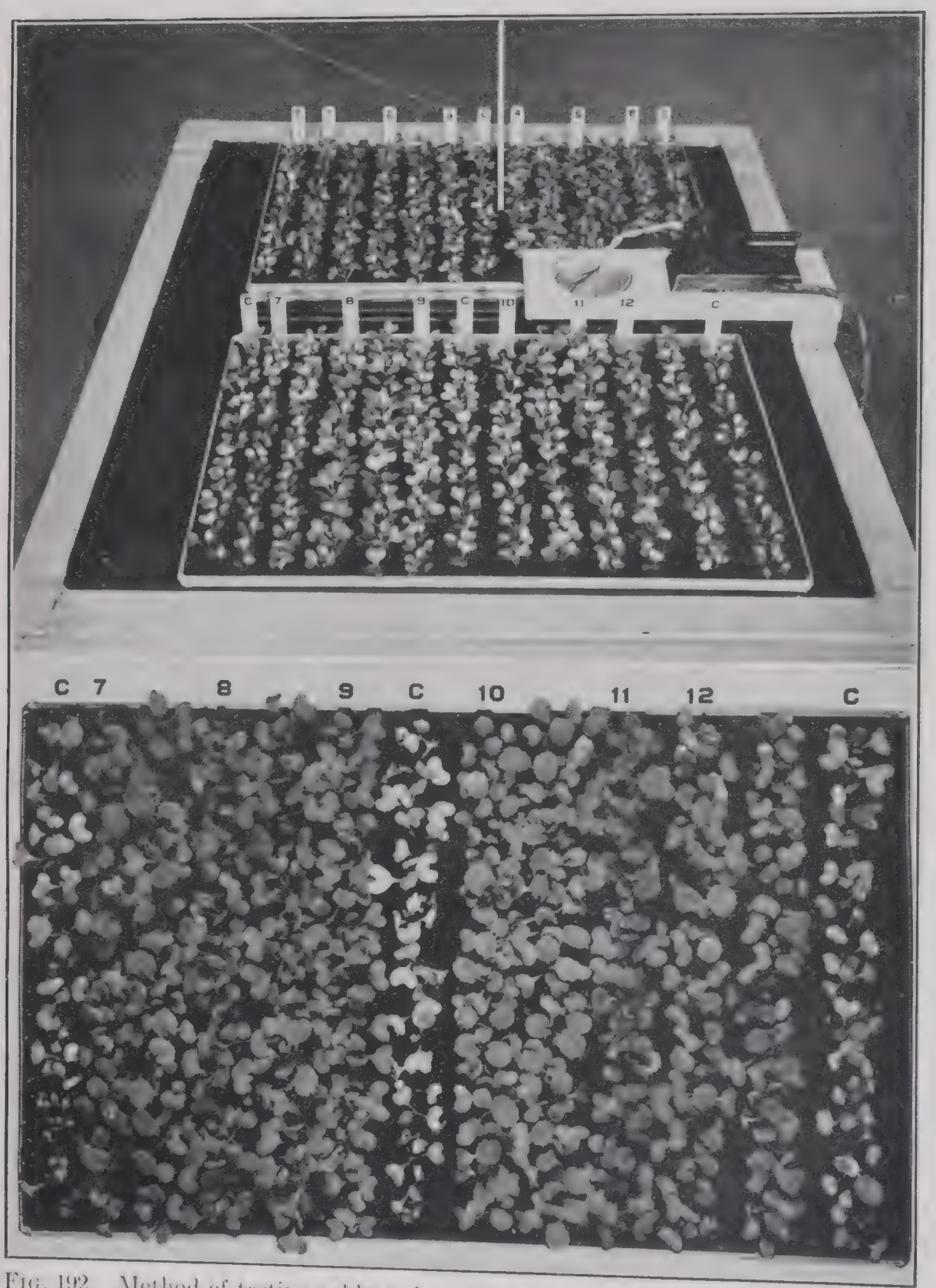


FIG. 192. Method of testing cabbage breeding progenies for resistance in constant-soil-temperature tanks. By growing seedlings in infested soil at 24°C., all plants showing the less desirable and less easily fixed type B resistance shown in Figs. 190 and 191 are eliminated. All seedlings which survive are homozygous or heterozygous for the dominant gene controlling type A resistance.

temperatures some varieties of wheat normally highly resistant to black stem rust (0, 1) gave susceptible reactions (3 or 4). In cabbage yellows (Figs. 190 to 192) and in pea wilt (17) monogenic resistance breaks down at high soil temperatures. Many similar cases might be cited in which temperature, light, moisture, or nutrition may alter the expression of resistance.

In other cases of monogenic resistance the range for disease expression is so narrow that, unless the optimum is maintained, many escapes will occur and expression of susceptibility is suppressed by the environment. This is sometimes the case with near-wilt of pea (25) and scab of cucumber (23). In the first instance natural conditions seldom provide a long enough warm period for near-wilt expression, and in the second instance sufficiently cool and moist periods are not prevalent in many localities.

As a rule, when resistance is polygenic in inheritance, it is relatively unstable in variable environment. In this respect it is similar to many quantitative characters. In the case of onion pink root, *Pyrenochaeta terrestris* (H. N. Hansen) Gorenz, J. C. Walker, & Larson, resistance is apparently polygenic. A variety resistant under field conditions may show various degrees of susceptibility as temperature and inoculum load are varied (9). In many studies with cereal rusts intermediate resistance, which is probabily polygenic, is more readily shifted by changes in nutrient, temperature, or light than is high resistance or high susceptibility (26). In carrying out breeding programs with this type of resistance, it becomes the more important to provide uniform testing conditions from season to season. Evaluation of breeding progenies is usually made in relation to standard resistant and susceptible tester lines in order to compensate for the variation in expression of resistance brought about by variation in environment.

METHODS OF DEVELOPING RESISTANT VARIETIES

The appearance of an individual plant or variety resistant to a given disease is usually only the beginning of control of the disease by this method. Resistance, to be effective in crop plants, must be combined with agronomic or horticultural characters of proved merit. In cross-pollinated plants such as cabbage, the objective may often be accomplished by selection within a variety which contains resistant segregates. In self-pollinated plants such as pea or bean, varieties are usually pretty well fixed for most desirable characters, the variants being rogues or undesirable off types. When resistance is found, it may be well fixed in a given variety and absent in others. Thus hybridization is necessary, and improvement is accomplished through backcrossing or through selection in successive generations. The procedure, thus, becomes the same as that

used for improvement of other characters in the crop concerned. Permanent progress in such a program depends upon applying proper technique in evaluating breeding progenies.

FIG. 193. Battery of Wisconsin soil-temperature tanks in use to select onion seedlings highly resistant to pink root, *Pyrenochaeta terrestris* (Hansen) Gorenz *et al.* Resistance in this case is polygenic, and temperature and inoculum are controlled to avoid eliminating all individuals and to retain only those which have a large complement of resistance genes (see Fig. 194). (*After Gorenz, Larson, and Walker.*)

The first requisite is having on hand or searching for individuals or lines of the host which show evidence of resistance. This may become a major task and require a long period of exploring and screening. When a resistant line is found, one may begin to cross with a susceptible but otherwise desirable type. In the meantime as much as possible should be determined about the reaction of resistance to the important environal

factors. If the pathogen is a soil organism, soil temperature, soil moisture, soil type, and soil reaction may be important. If the pathogen is a foliage invader, air temperature, humidity, and light are factors to be considered. A set of conditions may eventually be determined under which only the most resistant individuals survive. Such an environment is thus best for the evaluation of breeding progenies. The best means of applying inoculum is the next requirement. Examples of this are shown in the cases of onion pink root (Figs. 193, 194) and cabbage yellows (Figs. 190–192). In the case of the latter disease two types of resistance occur (p. 257); one is monogenic and very stable, while the other is polygenic

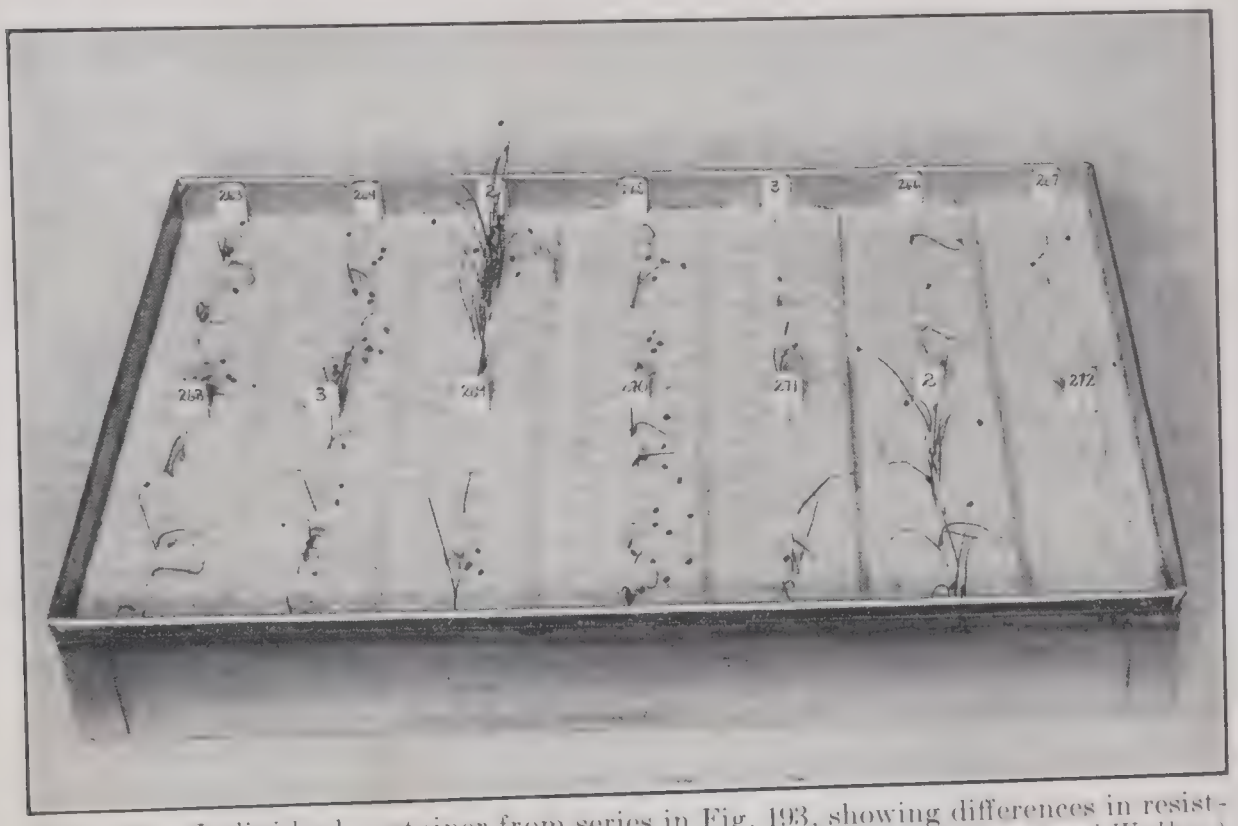


FIG. 194. Individual container from series in Fig. 193, showing differences in resistance of breeding progenies to onion pink root. (*After Gorenz, Larson, and Walker.*)

and relatively unstable. Under field conditions the two types are often phenotypically similar, the polygenic type breaking down somewhat during high midsummer temperature. Since both types may be present in the same progenies, the field environment is not a reliable one for elimination of plants which are resistant because of the less desirable polygenic character. By regulating soil temperature, it was found experimentally that polygenic resistance was ordinarily not expressed above 22°, while monogenic resistance was usually stable up to 26°C. Moreover, monogenic resistance is expressed in seedlings, and progenies can be tested shortly after the plants come above ground. By controlling soil temperature at 24° and testing in the seedling stage, monogenic resistant plants can

be distinguished from polygenic resistant and susceptible ones. Thus the desired individuals can be saved for further selection.

The above case is an example representative of very many in which controlled techniques have been worked out. If evaluation of resistance in breeding progenies is standardized, time-consuming operations often may be shortened simultaneously. In onion pink root the only resistance known is polygenic, and the most resistant lines may succumb completely if artificial environment is made too severe. By testing out various temperatures, a standard technique was developed whereby thousands of seedlings can be tested in a 28-day period in a relatively small amount of greenhouse space (9).

THE PLACE OF RESISTANCE IN DISEASE CONTROL

While it would be an ideal situation if we could look forward to having, eventually, disease-resistant varieties of all crop plants, it is undoubtedly too much to expect in view of the variability of some pathogens. The need of resistant varieties is greatest in the cases of diseases which do not lend themselves to other remedial measures. To be effective, resistance to a given disease must be combined with the currently desirable agronomic or horticultural characteristics of standard varieties, and with resistance which may have been established for one or more other diseases. Continual adjustment is therefore needed to meet changing crop requirements as well as changes in pathogenicity of the causal organism. Disease resistance, to be applied as a control program, therefore, is a continuous process which is most effective when coordinated with the general improvement of the crop concerned. The responsibility of plant pathology in plant improvement is indeed a large and important one. It should emphasize the continuous study of the pathogens concerned and their variability; their reaction to environment; the nature of host resistance, its heredity, and its reaction to environment; the improvement of testing and screening techniques, whereby resistance can be most readily and effectively integrated with a general plant-improvement program.

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